- - - - - - - - - - - - - - - -x
:
FIFRA SCIENTIFIC ADVISORY :
:
PANEL (SAP) OPEN MEETING :
:
- - - - - - - - - - - - - - - -x

OFFICE OF PESTICIDE PROGRAMS' PRELIMINARY EVALUATION OF THE NONDIETARY HAZARD AND EXPOSURE TO CHILDREN FROM CONTACT WITH CHROMATED COPPER ARSENATE (CCA)-TREATED WOOD PLAYGROUND STRUCTURES AND CCA-CONTAMINATED SOIL

October 23, 2001

[12:15 p.m.]

Sheraton Crystal City Hotel
1800 Jefferson Davis Highway
Arlington, Virginia 22201

P A R T I C I P A N T S

Stephen M. Roberts, Ph.D., FIFRA SAP Session Chair

Ms. Olga Odiott, M.S., Designated Federal Official

FIFRA Scientific Advisory Panel Members

Fumio Matsumura, Ph.D.

Mary Anna Thrall, D.V.M.

FQPA Science Review Board Members

John L. Adgate, Ph.D.

Michael Neville Bates, Ph.D.

James V. Bruckner, Ph.D.

Karen Chou, Ph.D.

Harvey Clewell, M.S.

M. Rony Francois, M.D., Ph.D.c

Natalie Freeman, Ph.D.

Gary L. Ginsberg, Ph.D.

Terri Gordon, Ph.D.

Steven Heeringa, Ph.D.

Nu-May Ruby Reed, Ph.D.

Claudia Hopenhayn-Rich, M.P.H, Ph.D.

FQPA Science Review Board Members

John Kissel, Ph.D.

Michael J. Kosnett, M.D., M.P.H.

Peter S.J. Lees, Ph.D., C.I.H.

Ross C. Leidy, Ph.D.

Peter D.M. MacDonald, D.Phil.

David W. Morry, Ph.D.

Paul Mushak, Ph.D.

Xianglin Shi, Ph.D.

Andrew Smith, SM, ScD.

Helena Solo-Gabriele, Ph.D., P.E.

Jacob J. Steinberg, M.D.

Miroslav Styblo, Ph.D.

John Wargo, Ph.D.

DR. ROBERTS: Thank you, Dr. Benson. Before we get to the questions, are there comments, suggestions, opinions, or remarks regarding this analysis until our discussion of Question 1 tomorrow?

Are there any questions of clarification only for Dr. Benson? Dr. Steinberg, Dr. Chou, Dr. Mushak, and then Dr. Ginsberg.

DR. STEINBERG: Yeah, I think we'll need an --

DR. ROBERTS: I'm sorry. Dr. Steinberg, you're going to have to use the microphone and identify yourself.

DR. STEINBERG: J. J. Steinberg. I think it would be important to have ATSDR tell us exactly what the basis of their justification of their uncertainty principal is, and I think that will be critical in answering Question 1.

RIGHT SIDE: Thank you. Dr. Chou.

DR. CHOU: Two questions. One, is there evidence to show children's metabolism is different from adults. You happen to hear lot of this work. I just want to know how much confidence you have in the first conclusion that there's no evidence to show children and adults are different. I mean to get to the point, just because there's no data out there, or you really think there's no difference.

And the second one is if you ever considered that some of the report of fibroepithelial thickening of arterial walls in children. I

don't known what's the background level in the general population, but these seems to be unusual to have this effect on blood vessels in young age.

DR. BENSON: Let me just -- in the work that we did, we did not look at the difference in metabolism. We were only looking at studies reporting adverse health effects. The metabolism studies were just not part of the evidence that we went through.

There are reports of enterepitheial thickening in major arteries in a couple of things reported. The most significant, or at least the most clear one, is from the result of our work in South America.

One of the South American publications dealt with the reported incidents of death in five children where the autopsy showed evidence of endothelial thickening of the walls. And I can't remember the organs now. But there were several organs involved. That report also is cited as Rosenberg in, I believe, 1974, where there's a detailed pathological report of those five cases.

DR. ROBERTS: Does that respond to your question, Dr. Chou?

DR. CHOU: Maybe. Do you really have confidence? How much confidence do you have to say there's no difference between children and adults?

DR. BENSON: You know, I guess that's a value judgment that everyone has to make for themselves. I was fairly confident when I went through the data base that there was no evidence supporting a difference in response between adults and children based on the data sets that are available. Other people have different views on that.

DR. ROBERTS: Thanks. Dr. Mushak, before we get to your question, I did sort of gloss over Dr. Steinberg. I didn't mean to gloss over Dr. Steinberg.

But if there is someone from ATSDR here who could briefly articulate their rationale for their uncertainty factors. Dr. Chen is here in the audience. If she could make her way to the table while Dr. Mushak is asking his question, then we can --

Dr. Mushak, why don't you go ahead and start.

DR. MUSHAK: Sure. Two quick questions, Dr. Benson. These are follow-ups on the questions of Dr. Chou.

One is have you been able at all to stratify this age band of zero to nine years into something smaller, number one. Number two --

DR. BENSON: Let me answer that one before we move on. Based on the publications, the answer is no.

DR. MUSHAK: Can you get the raw data?

DR. BENSON: Um --

DR. MUSHAK: I mean, this is a critical issue to getting the recall data.

DR. BENSON: That would probably be available from the Bazender (ph) study. I doubt whether anybody could reconstruct the same data back from 30 years ago.

DR. MUSHAK: Yeah. But there's a lot of data in those medical reports from Tseng that, you know, have been refined away.

That fact aside, what was the criterion for frequency of an effect before it became important, say, neurological versus skin? Obviously, the longer the age band for the so-called child age, the more skin is going to rise in ascendancy and the less the neurological. I mean, I'm bothered by this. And I think it needs a clarification. What voted an effect in and what voted an effect out?

DR. BENSON: It was primarily what was in the reports. We didn't try to second guess the authors of the publication. If they said there was an effect there, we took that at face value.

DR. MUSHAK: But in terms of the frequent quantitative, I mean, that's the question that remains on that, too.

DR. BENSON: Yeah. And what I'm going to say is, again, we relied on the call from the investigator as to whether it was a significant effect. We did not have criteria that we developed

independent of what was in the publicly.

DR. MUSHAK: I'm bothered by judgments that are based on what an author says.

DR. ROBERTS: Let's move on to the next question.

DR. MUSHAK: Sure.

DR. ROBERTS: Dr. Ginsberg.

DR. GINSBERG: I just want to make sure that this is explicit. It's implicit in one statement you made. I assume that in the various exposure assessments that formed the basis of this dose response and the epi studies that they did take into account food exposure and other environmental background exposures as part of your dose.

DR. BENSON: Some of the studies included an estimate from food. And when that was in the publication, we used what the author said was the exposure with whatever assumptions we need primarily in body weight to get --

DR. GINSBERG: The big factor in arsenic areas is you can have fruits from rice to soups to everything being contaminated. So it would be a vanguard to dose that.

DR. BENSON: The Zolovar publications did take into account, at least tried to take into account, exposures from food. I've got some doubts about how accurately that was done.

Most of the studies do not include other environmental exposures. EPA tried to add into the exposure from the same study an estimate from food. The rest was only either drinking water or soy sauce or whatever the publication was primarily reporting on.

DR. ROBERTS: Dr. Chen, do you have a response for Dr. Steinberg?

DR. CHEN: First of all, I'd like to thank EPA for inviting me here. I'd like to have a chance to clarify that, first of all, MRL, minimal risk level, is used as a screen level. We may be talking about different things. We're taking about actual levels. It's not an actual. It's not a cleanup level. It's just designed to be used as a screen level for health assessors who just select contaminants of concern and to weigh sites.

So that having been said, therefore, our numbers tend to be sometimes more than EPAs levels. A lot of times they were the same.

But in terms of the acute oral minimal risk levels that we have derived, it's a provisional number. It's not what we considered a full-flash, kosher MRL. MRLs are used as a screen level; and, therefore, the methodology caused the deriving number based on less serious health endpoints rather than serious effects.

Looking at the acute or acute data base for getting arsenic, the

data is very limited. And mostly they're all poisoning cases, you know, a lot of fatalities and so forth.

At the request that we originally received from EPA, we're asked to come up with a number. Therefore, we had to strive real hard. The only thing that we could come up with was the Tsuda study which is the best you can find under the circumstances.

Still the health effects were considered and have been alluded to many times, especially with the nonreversal neural path peripheral neuropathy. I think that was reported as considered serious. And so, therefore, under our normal circumstances, we have not even derived a number since we have to we call it provisional.

The low level milligram per kilogram per day is based on the authors assertion three mil per day of soy sauce ingestion and the body weight of 55 kilograms for the Asian population, which is Japanese, and the dose. There's no problem with dose.

And so the factor we used was 10 because we cannot see that if we were to use 3, as EPA originally. Or finally, you know, we had to -- we had to decide on the 10. If we were to use 3, that would be considered as a minimal MRL.

There's no way -- none of the local members, Dr. Benson here, EPA representative on our MRL working group meetings and

participate fully. And we had a lot of discussions, and it's just very, very hard for all of us to agree that those facts can be considered as minimal or less serious. So we had to use the 10.

And we did not use the fact for interhuman variability. We assumed the data base included different ethnicities, including children. That was our rationale. We used a 10.

DR. ROBERTS: Thank you. Let me jump in with a question, and then Dr. Kosnett and then Dr. Ginsberg.

Dr. Benson, there was something you pointed out in the first part of your presentation that confused me the little bit, so I'm going to ask the EPA to respond because I think they're the ones who can -- OPP -- that can clarify this.

You talked about that this is really developing a value, if I'm not mistaken, for intermediate exposure which is defined by OPP as one to six months. I mean, they've got lots of different descriptions about what periods these apply to.

In the initial presentation, I thought for this particular scenario we're looking at six years. So I guess my question is: How do toxicity values for these shorter periods of time fit into your assessment for an exposure scenario that involves six years of exposure? Or have I misinterpreted something?

DR. MCMAEHON: Well, I'll try and clarify that for you.

Aside from the way the durations are expressed between agencies, our values, as I said before, we try to match up the endpoint values with the temporal characteristics to the exposure. So by our definition of a short-term or immediate-term exposure, we want to have values from data that you've already seen that kind of match with what the duration of exposure was, in this case human populations for arsenic.

Longer term exposure, as I said, I didn't show them; but there are some published values. And you did see some of the data from Dr. Benson from the Tseng study with the NOAEL value from the chronic exposure. We kind of go along those lines to get endpoints that will be characteristic of different types of exposure.

Did that clarify for you or --

DR. ROBERTS: Well, maybe we can talk about it some more when we talk about the exposure assumptions that you're going to use in the assessment and how they might match up. So that's fine. Let me go ahead and ask Dr. Kosnett for his question and then Dr. Ginsberg.

DR. KOSNETT: Dr. Benson, I wanted to ask you, just to see if I followed correctly, how you estimated that no-effect level for skin

lesions in children could be 0.03 milligrams per day for up to 10 years of exposure based on the Tseng study.

I'm just going to walk through very quickly what I think you did, and I just want to make sure that we're on the same page.

Basically, EPA assumed 4.5 liters consumption in a 55 kilogram adult male.

DR. BENSON: That's correct. Yeah, for the Tseng study, it was an estimated value of four and a half liters per day of water consumption. The Sevrion study actually had reported water consumption in the population.

DR. KOSNETT: The Tseng study didn't say that. That's EPA estimate.

DR. BENSON: Correct. Yes, that's correct.

DR. KOSNETT: And then you multiply --

DR. BENSON: So that comes out to -- if you divide 4.5 liters per 55 kilograms, it comes out to 82 milliliters per kilogram. And then you multiply that times 1.9 to get from an adult to a child.

Yes. The average exposure that EPA was using in the Tseng study was 0.014 milligrams per kilogram body weight per day which was in turn derived from the concentration of water in the wells and the assumption of four and a half liters per day for drinking water

consumption. And, basically, I multiply that value by 1.9 to correct for children.

DR. ROBERTS: Thank you. Dr. Ginsberg.

DR. GINSBERG: Regarding the NOAEL in children for the skin, it looked like the epidemiology that you're relying on focused on skin lesions. And I can imagine in these large populations studies that that would be a good easy endpoint to get.

But I'm concerned that neurological endpoints, especially subtle neurological endpoints, may not have been looked at in these children. I haven't read these studies, but I'd like your comment on how much confidence we should have that, in fact, would be a representative NOAEL in a young child with a developing nervous system in terms of what these studies actually looked at.

DR. BENSON: That's a very good question, I think.

If I remember correctly, the Tseng study really only focused on skin lesions. I don't think they had any evidence in the -- there's no evidence in the written publications that they looked at neurological effects at all in the children.

I think the Mazuta study that was done somewhat later, I don't -- there's nothing in there that I recall as focusing on neurological effects. Whether that was a conscious admission on the part of the

authors or whether they did not look at all, I can't tell. Someone who is more familiar with the list of authors on the publications could, I think, provide that information. But I have not quizzed the authors myself.

DR. GINSBERG: So then the natural follow-up question is: If we don't have the neurologic data from the Epi studies, is there any data, either in animals or humans, acute or longer term, that suggest that in young children the skin endpoint is a good surrogate from the neurologic endpoint?

DR. BENSON: There's nothing conclusive on that that I'm aware of. Most of the studies of large scale populations, I don't think, looked carefully at the correlation between skin lesions and other symptomology in the way that you're asking.

DR. ROBERTS: Other questions from the panel?

Okay. We're deciding -- we're caucusing on the agenda. We're at the point of the day when we were originally scheduled to break for lunch.

Dr. Abernathy, I don't known whether you want to be in the unenviable position of being the last speaker before lunch or the first speaker right after lunch.

DR. ABERNATHY: Well, why don't I go right after lunch.

Everybody will be asleep.

DR. ROBERTS: With that recommendation --

DR. ABERNATHY: Whatever you want. It could be either way.

DR. ROBERTS: It's about a 20-minute presentation; is that right?

DR. ABERNATHY: Yes.

DR. ROBERTS: Let's go ahead and break for lunch. Is that all right with you, Dr. Abernathy?

DR. ABERNATHY: Fine.

DR. ROBERTS: Would you be available to present it right after lunch?

DR. ABERNATHY: Yes.

DR. ROBERTS: Let's take a break for lunch. Let's convene sharply at 1:30 and begin.

[Lunch break. Conference resumed at 1:30 p.m.]

DR. ROBERTS: I think we have a quorum from the Panel back from lunch.

I would like to thank Dr. Abernathy for agreeing to delay his presentation until after lunch, but I think we're ready for that

presentation now.

Dr. Abernathy, are you ready to go?

DR. ABERNATHY: Yes.

DR. ROBERTS: All right.

Then the next item on our agenda will be an assessment, or I guess an update, on the review and status of arsenic regulation in EPA's Office of Water; and that will be presented by Dr. Charles Abernathy.

DR. ABERNATHY: Thank you very much. It's nice to be here, I guess. But I do want to thank Steve for extending it after lunch. He said if we went past 1:00, OPP was going to pay me overtime.

Ma'am, the next slide, please.

The reason you see this put down this way is water is changing so fast it's kind of hard to make beautiful slides. My last group of them I had to change all of them.

What I'd like to do is show you what we do at the Office of Water. I think this is probably puzzling to some of you. Don't feel bad, at times it's puzzling to me.

What I'd like to do is give you an overview of the statutory requirements. And I'd like for you to remember that the FIFRA law and Safe Drinking Water Act are two different things. What we do is

governed by the Safe Drinking Water Act. And some of the things that are required for us to do are not required by other people; and what they're required to do, we are not required to do.

So in that context, I want to give you the statutory requirements of what we do so you'll have some way. Then I'd like to show you how we develop our standard. You'll find out here. Then I'd like to look at the exposure we've looked, the health effects. And then look at things that most people don't use as Safe Drinking Water Act specifically says you'll at a PQL, which is a practical quantitation limit. You will, also, calculate in costs and benefits. And then where we hope we're going, and we're going somewhere.

All right. Next slide please.

Why did we develop a new standard? Well, the old standard 50 ppb was set roughly 60 years ago. We aren't changing it because it's 60 years old. I'm almost 60, so I hope that's not the reason. But we were using old science. There's been a lot of new science coming out so that's the reason we're changing it.

The '86 Safe Drinking Water Act said we had to set a new standard by '89. Anybody familiar with arsenic, there were about 300 lawsuits on both sides and from the good guys, the bad guys, and everybody in between. So we didn't meet that deadline.

'96 Safe Drinking Water Act, they said we had to propose by January 2000 and final by the first of 2001. We were only a month and a half late on the proposal. We got it out February 22, 2001. All of us went out, and those that drank alcohol had a lot it. Then we woke up and found out we had to redo it.

So what we did to redo it is we had a National Academy of Science and Science Advisory Board as we were looking at it, and they both said recommend a downward revision as promptly as possible. So that's what we focused on. Next slide.

We have a process for setting. We have two parts of the Safe Drinking Water Act. We have an MCLG, which is a maximum contaminant level goal. This is a health goal. It may or may be reached. It's where we would like to be if it was a perfect world. Since it's not a perfect world, we also have a maximum contaminant level. This is the enforceable part of the Safe Drinking Water Act.

When you see a MCLG, it could be any number. That's where we'd like to be. For example, with linear carcinogens, it's usually zero; has been in the past. This is subject to change, but we've always done it that way in the past. With the possibility of motive action data, it's possible we may have a greater than zero for a carcinogen.

The maximum contaminant level would then be set as close to

the MCLG as is feasible. And then once we look at that, we look at benefits, and do they justify the cost. We go directly to the feasible level. If they did not, then we would he consider raising the MCL. This would be part of the risk assessment. Next slide.

What we've done and what congress requires is that we look at the peer-reviewed research. This is research that's been published. We do a risk assessment, where the part I work in is the hazard identification. That's not difficult with arsenic. There's enough hazards associated with exposure to arsenic. We look at the dose response, which is always questioned and everybody has different ways to interpret it; and then we look at the exposure. We come up with an MCLG and a risk characterization.

We would then -- on the risk characterization, we tell our management how well we think the various parts fit together, how strong each parts are, do we have, as with some epidemiology studies, not a good exposure assessment. So we would say the exposure assessment is weak. But the hazard identification, for example, of the Tseng study, the cancer, the skin cancer, was very strong.

And then the risk assessment part, and this is what the management does. They look at the treatment costs of small system technologies, test methods, costs and benefits and occurrence in the

number of systems. Next slide.

If we look at exposure, one of the things in the Office of Water, since the methodology is total arsenic, is we look at total arsenic. And that's the way the rules are written. However, in drinking water, you primarily have Arsenic III and Arsenic V.

There are many forms in the environment. We've mentioned some of them today. Other forms that we're not as concerned with are some organic metabolisms. And I'll get that.

How does it get there? In places like Fallon, Nevada, weather of rocks and other places, surface water mining as the water runs off. We have two types of methylated species as I call them. One is those that are methylated inside the body. So there we have the monomethyl and the dimethyl arsenic acids. They both occur in the +3 and the +5 species.

It appears from the data we have right now, the +3 species of the monomethyl and the dimethyl are toxic. Whether they are the punitive toxic agents is not settled at the present time.

In food you have a lot of organic. We need a lot more data on this. In fact, with Pellizzari in North Carolina we're actually looking at the forms of organic arsenic in food as well as inorganic. However, if you look at fish and seafood, it's largely arsenobetain, which is

absorbed and excreted as the parent compound not broken down. It appears that that's pretty safe, so you can continue to eat fish and shrimp if you like. Next slide, please.

From some data with U.S., we took the market basket survey. And I will say that this is for the entire United States. It's not for any individual area.

Then we looked at what data we had on the speciation of arsenic in various foods. This always takes a little bit of a risk. Because if you're growing in different parts of the country with different soils, you're never sure that the level of arsenic is exactly the same or what form. But it was the best estimate we could make at the time. And that's why we're doing the Pellizzari.

But the intake average in the United States as a whole was approximately 50 micrograms per day. Of that, approximately 10 micrograms was inorganic arsenic.

If you look at Taiwan, there's only been one study. They have an average in the range of 50 micrograms of inorganic arsenic per day. This study needs to be repeated. But you know, exposure could vary quite markedly in your food. Next slide, please.

Hazard. This is what I'm talking about. There's no absence of effects. You need to pick out of the ones that you should pick out.

All the early work was done on skin. Then there's bladder, lung, liver, kidney, prostate. At the present time, EPA is looking at bladder and lung -- the Office of Water -- for quantitation purposes. The data is better.

Chronic. You have skin lesions, vascular obstructive lung disease, and diabetes. At the present time, we are looking at quantitating from a cost benefit standpoint the vascular and the diabetes. These are the ones we seem to have the best data at the present time for.

Animal Affects. There have been developmental reproductive proposed. They've always been at high doses.

Cancer. This is the only human carcinogen we know of in which there's absolutely no reliable cancer model in animals. There have been a few reports. The model from Australia, but there are problems with the modeling. Next slide.

Mode of Action. If we look at the early reports, inorganic arsenic was not directly mutagenic. However, it was codomutagenic. If you put arsenite and UV together, you got a greater effect than with the UV only.

It does have definite reflex and effects on DNA repair. Both the NRC -- and this was the 2000 -- and the 2000 EPA panels concluded

that the dose response associated would be sublinear or threshold in shape.

Recently, some organic metabolites are DNA reactive and affect gene expression. So we're reexamining that. Next.

This is what we're doing. This we quite admit we stole it directly from Louise Ryan because she had such a beautiful graph. But by and large, we looked at it. With arsenic, you're very fortunate because you have a human population. And with most of these, when we look at it, we look at a 5- or a 10-percent level for an effective dose. But in this case, with the large human population, we go down to an ECO_1 or an effective dose for 1 percent.

We then calculate the 95-percent confidence limit on the lower bound. That becomes our LAD_{01}. And the question earlier about variation in exposure, this is one of the ways we try to take care of it by looking at the 95-percent confidence limit on the maximum likelihood exposure.

Then we would do one of two things. We would draw a straight line to zero with a ruler and say this is the best we can do at the present time. We don't know what the shape of the curve is underneath the 350 on the LAD_{01}. You could draw theoretical lines for it if you thought it was sublinear.

We have done the same thing with lung cancer. And this type of work is what we're using to quantitate the costs and benefits from reducing the exposure to arsenic. Next one.

New Studies. I mentioned new studies. Mark Mass, et al., in 2000, the Chemical Research Toxicology, has some data that a metabolite, the MMA3, for example, DMA3, may break DNA. So you could have the possibility of a direct interaction. Therefore, we would certainly not use a sublinear from that standpoint.

There's a new study in New Hampshire, Dartmouth, on skin cancer instance in high arsenic. We're also looking at that to look at arsenic effects of rural water in the United States.

A lot of the criticisms in the Office of Water have been you have no U.S. studies. Well, that's probably true. We have a lot of U.S. studies. They're just not very big. And people say we don't care what happens somewhere else. We want a study in the U.S. So we have to answer that.

But this is one study that we are looking at that was actually done in the United States. Next slide.

The NRC Update. We are actually quantitating. This is 2001, the one that just came out. We're actually quantitating the bladder and lung cancers. And they said those should be the focus of our risk

assessment. They say the Southwestern Taiwan data are still the most appropriate for risk assessment and that the present mode of action are not sufficient to depart from the default assumption of linearity. Which means that when we come to the LAD01, we would draw a straight line to zero to do our calculations. Next slide.

This is just a set of numbers, and this is some of the ones we're looking at. And this is just for illustration purposes. We're not sure we're going to use this model. But it just shows you, if you look at the MLE and the excess lifetime risk at these values, you can see that from 3 to 20, you go with female bladder cancer 4 to 24 and 7 to 45. So as you go up, you're going to allow more bladder cancer. This is going to be balanced by the cost of treatment.

VOICE: Is that for 10,000?

DR. ABERNATHY: Yeah. Next slide, please.

This is something I just want to touch on, and this is where we differ. We can come to the same point in the road as our colleagues at OPP, for example. Just assume we did.

But here is where we will diverge from other programs because we have to look at a practical quantitation limit. In this case, it's three micrograms per litre. Our practical quantitation limit isn't what can be done in a university lab. It's what can be done in a contract lab

within a reasonable cost.

Because when we put out a regulation, they have to sample so many times a year and send the results to us. So we can't require them to go down to .1 microgram because it would cost them too much money and could not be done easily. So that would be the low point or the baseline for the arsenic occurrence -- I mean, excuse me, the arsenic level in water we could say would be three would be the lowest.

Then we would look at our occurrence date. We have our own data. We have that from others. They agree. Then this would help us in our calculations of cost.

We have a certain amount of treatment. Obviously, if you're going to treat for large and small systems, this is very important. For example, if it costs the City of Los Angeles $8 million to treat for arsenic, well, that's not really very much money for 8 million people in Los Angeles. If it costs $100,000 for a group of 25 people, that's a lot of money.

So we look at both large systems, and we also look at small systems because sometimes the economic impact -- for example, they've done calculations for a large system. You're talking about pennies per month on your bill. If you look at small systems, you're

talking about anywhere, depending on your calculations, up to $200 a month, $150. So we do those calculations.

And we do that for both benefits for cancer, noncancer. And we also have a section on affordability for small systems. And these all go into the overall final number. Next slide.

What procedural steps are we going to take? Well, in 2001, I think most people are familiar, we actually put a 60-day extension on April that was extended nine months until February 22, 2002. And we finalized this extension. That means that the new arsenic regulation should be out February 22, 2002, which is kind of nice because on February 20, I'll have 29 years in and be eligible to resign; and then I won't have to answer those questions that our good friends send in.

To give you an example -- and that's one thing I think we ought to mention -- is that we do answer all questions. For the other arsenic rule when we proposed it in February of this year, there were over a thousand questions submitted for us to answer. And we did answer every single one of them.

In the near future, we will really propose a rule soliciting comments. We hope this will be in the middle of November on 3, 5, 10, and 20. As I mention, we can go no lower than three, because that is our practical quantitation limit.

In May, August, we'll be seeking outside expert review. Next slide, please.

We've had three of these when had the National Academy of Science do their update. And the final report, Arsenic in Drinking Water 2001, is available at the web site. You have a copy of this. I will say I haven't been to this web site, but people who have said you can only download one page at a time. If that's true, anybody that's got a graduate student has a good project for them.

But they have given, as I mentioned there, reasons for it that we should quantitate lung and bladder, that the Southwest Taiwan is still the best data, and that there's no reason at the present time to depart from linear. Next one.

In addition, something that a lot of people don't know about. We looked at the cost. NDWAC, which is the National Drinking Water Advisory Committee subgroup. These people are mostly engineers. I went to this meeting; didn't understand a word they said. But basically, they said that our cost estimates were at least reasonable.

This one is you can get on the web site. You have the www.epa.gov.safewater. And you can download this one. This is an EPA document and is for anybody who wants to download it to look at

it. Next slide.

The Benefits. We had a science advisory board, your counterparts over in water -- we call it the science advisory board -- in which we had a bunch of economists and a few toxicologists. And they got together and came out with a report. And what they asked us to do was look at total benefits and cost, incremental, and things we hadn't been looking at in the past.

One of them is latency, which is how long after you've been first exposed to an effect. Well, some people, mainly OMB, really wanted to look at that because it cuts down the cost. However, we also decided to look at the other side of that, and that's recovery after cessation of exposure, which is another important part of it. And right at the present time, we're using smoking as just a guideline because we don't have good enough data at the present time on arsenic.

This report is also available on the EPA web site. If you're interested in what they said, you can certainly download it. Next side.

Well, The Next Steps. And just to go through very quickly because I'm sure that you probably aren't interested. We have a lot of legal regulatory policy and the scientific considerations that have to be done. We're incorporating right now the results of all three expert

panels into our cost benefits and MCLG, the health endpoint. We'll have another opportunity for the public to make comments. And we'll make a decision and publish it around February 22, 2002. Thank you very much.

DR. ROBERTS: Thank you, Dr. Abernathy. Are there any questions from the Panel on the update on the regulation status? Yes, Dr. Solo-Gabriele.

DR. SOLO-GABRIELE: I had a question about the low level, 3 micrograms per liter. Was there a cost analysis, a cost benefit analysis, done on that?

DR. ABERNATHY: No, no.

DR. SOLO-GABRIELE: It was just based on the laboratory --

DR. ABERNATHY: What it's based on is the EPA has its on laboratories for analysis. And we do them and we send them to various contract labs. And these contract labs are small labs that actually do a lot of analysis for water systems, among other water systems. And they have their methodology that they can do. And these range $10 to $50 in general. If it goes over that, they say it's getting out of a practical quantitation limit just due simply to cost because we're talking about arsenic here.

But this isn't the only thing they have to analyze where they

have a whole list of analyze. So you really are in what can be analyzed for relatively inexpensively and quickly and in a group. So all those factors roll in.

We presented this data to the Science Advisory Board, and they agreed with the 3. They felt that was where it should be. And the other thing is it will go down in the future. But that is the one right now.

DR. ROBERTS: Any other questions? Yes, Dr. Smith.

DR. SMITH: Thank you. I've only have had a chance to look briefly at the update, the NES update. And my recollection -- perhaps some of the epidemiologists and others involved with the committee at the table can help me. There appeared to be some discussion about different approaches when you're doing cancer risk estimates for whether you use baseline cancer incidents associated with the United States versus Taiwan.

Is EPA carving out a position of where they're going to come down on that?

DR. ABERNATHY: At present time, I'm not sure. I -- just let me make -- a few of these things are still under discussion, and they're internal Agency, you know, EPA matters. And a decision hasn't been made. So I'm not positive yet on that one. But they're doing the

calculations on both ways.

So until whoever, and it's certainly not me, makes that decision, and it's made public, I don't know. But it's a good question, and I'm not sure which one they're going to do.

DR. ROBERTS: Any other questions? Dr. Matsumura.

DR. MATSUMURA: Well, I suppose we need clarification that the particular committee should really think about the economics for any of those policy questions and what not. This is just for ourselves. I know this is not the drinking water. But some of those exposure modes may come close, and this particular presentation had the cost and that type of considerations. So it is something that we have to discuss whether we stick to science, or we're going to have some consideration on economics. We have no economists here.

DR. ROBERTS: Are there any other questions for Dr. Abernathy? Yes, Dr. Clewell.

DR. CLEWELL: Can I ask a question that's actually for the people in the pesticide office relating to what he just said? How do you intend to use the work that they're doing for the MCL because I presume you don't have to balance cost and benefits in the pesticide office like they do for the regulations regarding drinking water. Are you planning to use their risk estimates and then use your own

policies regarding acceptable cancer risk?

VOICE: I'll try to answer that. We do hope to use their approach, the quantitative modeling approach, to the assessment of the risk. But probably wouldn't start using -- you know, they've already got water numbers. So we don't have to do that. It would just be related to the treated-wood exposures.

But, you know, that information was considered updated or, you know, to take into serious consideration compared to the linear default that's published in the IRIS data base. That's basically how we would use it.

DR. ROBERTS: Are there any other questions? If not, thanks very much, Dr. Abernathy, for you update on events.

Before we get to the public comments, I would like to make you aware that our final member of the Panel has just joined us, Dr. Wargo. And not to put you on the spot right off the bat, but we did a little initial thing where everyone introduced themselves, their name, affiliation, and their expertise. If you wouldn't mind, can you sort of fill us in.

DR. WARGO: Sure. My name is John Wargo. I'm a professor of Risk Analysis and Environmental Policy at Yale University where I've been for about 15 years. I specialize on kids's exposure to toxic

substances. And I also float into the legal arena.

DR. ROBERTS: Great. Thanks very much.

We now come to the point on our agenda where we take comments from the public. And this is a very important part of the meeting because this gives us our opportunity to get a variety of other perspectives on the various areas that we're going to address. But let me just make a couple of announcements before we start the public comments.

One is that I would ask each public commentor to stick to their allotted time. We have a lot of people on the list that want to comment; and in fairness to them and to allow the Panel time to deliberate these issues, we need to make sure that everybody sticks to their allotted time.

Also, there are a lot of issues associated with CCA air-pressure-treated lumber. So potentially there are lots of points that could be made in the broad universe of things. But what we're meeting here to talk about are the scientific issues associated with a specific preliminary analysis by the EPA.

So I would like each of the public commentors please to confine their comments to scientific issues that are germane to our discussion and germane to this Panel rather than making broad statements about

other CCA issues.

As each public commentor's turn comes -- and we're going to take them in the order in which we've received requests to address the Panel. I mean, there may be some logical sequence to the presentations. But not knowing in advance what each person is going to say, we have no way of knowing what that is. So we're just going to take them in the order in which people have requested the opportunity to speak to the Panel.

There is a place. It's right up in this corner of the table, right next to where Dr. Abernathy was, that's designated for the public commentor. Just come forward, sit down, introduce yourself. Let us know your name, your affiliation, and who you represent. And then go ahead and give you your comments.

I would, also, ask that you would be available immediately after you give your comments to answer any questions or clarifications that the Panel might have for you.

Again, I apologize. We have to stick to a fairly tight schedule because we have a lot of commentors. It would be -- ideally, we could engage in some discussion and dialogue with each of the public commentor, but we really don't have the opportunity to do that, unfortunately.

So, please, just take this time available to emphasize your main points. For folks that have submitted materials in writing, those have been distributed to the Panel. That's the best venue, frankly, to get the sort of detailed technical information.

As we begin, we're going to take them in order. And I'll sort of announce who's up and who's on deck to kind of keep things moving along. If you're going to be the next one up, if you could start working your way up to this part of the room so you can jump in when your turn comes.

The first individual that's on our list is Mike McGrath, and he will be followed by Jane Houlihan. Is Mike McGrath present in the audience? Okay. Then let's go to Jane Houlihan, who'll be followed by Chris Williams.

MS. HOULIHAN: Let's get our presentation booted up here just to let you know in a nutshell what we'll talk about today at Environment Working Group. We're a public interest research organization, nonprofit --

DR. ROBERTS: I'm sorry.

MS. HOULIHAN: -- based in Washington.

DR. ROBERTS: I'm sorry to interrupt you right off the bat, but can you introduce yourself for the record.

MS. HOULIHAN: Jane Houlihan. I'm Research Director at Environmental Working Group. We're a nonprofit public interest research based in Washington, D.C. And we've spent the last several months putting together data on arsenic-treated wood for an exposure assessment that we'd like to present to you today.

We've done a Monte Carlo-style risk assessment to look at the full range of risks that children might face from exposures to play structures and decks built from arsenic-treated wood.

I, also, would like to acknowledge my coauthors here, Sean Gray and Richard Wiles, at Environment Working Group. We put this study together, the three of us.

So just to start out, just as a basic reality check, I just wanted to remind people who aren't in regular contact with toddlers -- if you could go back one, Sean -- how kids play on pressure-treated wood just to remind ourselves. In the end, it all comes down to numbers.

But when you look at it on the playground, these kids really do contact quite a bit of the wood. You can see in this picture two little boys on the ramp are, lying down on the ramp. They have short sleeves and shorts on. Two little girls have their bodies against the wood posts. This is all pressure-treated wood. Toddlers also mouth the wood and rub on the wood. Things we never do as adults are just

perfectly normal behavior for little children so the exposures are quite different from adult exposures. Okay. Next slide.

Basically, our risk assessments that have been done to date, and there have been several by the Maine Department of Health, California DHS, and the University of Florida and CPSC's 1990 assessment.

Those assessments done to date have been point estimates of risk that have looked either at an average expose or some sort of reasonable upper bound exposure. And ours is different in that we've simulated in a Monte Carlo-style assessment what might be a more full range of risks from the low end to the high end given the range of children's body weights and the style of play that children have.

And if you can -- you should have a copy of this presentation. If you look under the explanation of our scenario, basically, the run that I'll present today, we've looked at a million children in this run. We simulate their play from ages one through six years of age up to their seventh birthday. And we, also, focus on the subset of kids that you would be most concerned about. Those are kids who play fairly regularly on pressure-treated wood.

One group of children we look at we assume plays three times a week on the wood. And then we add on a second group of kids

assuming that they have a deck on their house and they play maybe three times a week on the deck at their house.

This model, we -- the things that are variable in our model that make it a Monte Carlo-style risk assessment are listed on the left-hand chart there. We vary body weight and surface area. So for each child that's run through the model, we choose a body weight and then calculate a surface area based on measured values.

We allow the range of arsenic concentrations in contaminated soil beneath the play structure to vary. And when I say "vary," the variability is still all based on measured distributions from studies that have been compiled by EPA in this process. They have copies of all these studies, I believe, that we've used in our risk assessment.

We, also, let vary the dislodgeable arsenic that adheres to a child's hand and skin, also based on the many studies that are available for that parameter.

And then lastly, you know, the question of how much soil do children ingest daily. We have high -- in that exposure parameter, we have high-, medium-, and low-exposure children and allow ingestion to vary within each of those categories.

And all other model parameters in this Monte Carlo assessment are fixed for the simulations. So we use, you know, for a

bioavailability, for the amount of time that children play outside, for soil adherence to skin, all the other standard parameters that go into these kinds of models. We use the parameters proposed by EPA in their document that they prepared for this meeting.

I, also, compared that to a study done by Gradient, funded by Osmose and Arch Chemical Companies, to see what sort of range the spectrum looks like in different assumptions that people choose. Okay. Next slide.

This data is incorporated into our model. These are the three studies that were available to us that looked at the amount of arsenic that rubs off onto hands. So this is actually data from hand-wipe studies where normally an adult volunteer goes to a deck or a play structure, rubs their hands on the structure, and then it's rinsed off and measured in the laboratory. It's done on a surface-area basis.

So in the interim, these studies, you have numbers that are in micrograms per hundred square centimeters of hand in this case. In hand area, it's normally the palm area.

One of these studies was conducted by the State of California. That's an adult hand on a municipal play structure. The middle group of triangles there represents the data from the Maine DHS Study in 1998. That was an adult volunteer, wet and dry hands. This volunteer

rubs their hands on a deck that was three years old. And you see a big variability depending on the conditions of the hand and the rubbing style.

And the third group, the triangles on the end, is a wood industry study conducted by SCS in 1998. And that study focused primarily on new sealed wood. It looked at dislodgeable arsenic that ended up on the hands of adult volunteers. And you can see the residues are lower on that study because most of the wood from that study is sealed and dislodgeable arsenic is lower on that surface of the new one. Okay. Next slide, please.

And this is the other half of what goes into our equation for how much arsenic would end up on a child's hands. Each of these sort of vertical lines represents an individual study of dislodgeable arsenic on a particular structure. And this is arsenic that ends up on a wipe. So all of these are wipe samples. Some of these are wet wipes; some are dry wipes. They are conducted by -- each of the legend on the right-hand side is in order for how the dots progress across the chart.

And the Y axis here is in micrograms of dislodgeable arsenic per hundred square centimeters of wood. So you can see it's pretty variable. These studies are dominated by new sealed wood.

There's a few of the studies, sort of the middle grouping of

studies, represent 14 play structures that have been sampled by the State of Connecticut and by the California Health and Welfare California Study. So those 14 play structures were all aged and heavily trafficked and have sort of moderate dislodgeable arsenic concentrations.

The third group of samples from the left, which is the pink samples, represent samples that we've collected over the past several months. New woods. Most of it is unsealed, purchased from retail, decking boards, two by fours. So this is what you would buy at a major retail store if you were a home owner.

And we sampled this wood with wet wipe methods and got, in some cases, dislodgeable arsenic concentrations as high as a thousand milligrams in our extreme sample per 100 square centimeters -- I'm sorry -- micrograms not milligrams -- per 100 square centimeters of wood.

They can do a quick -- this is a really important graph in my mind because it can be quickly compared to the cancer risk, excess lifetime cancer risk, computed by NRC for drinking water.

In the NRC update, essentially, three micrograms of arsenic per liter of water ingested daily at about a liter a day corresponds to a one-in-a-thousand cancer risk. So if you look at this graph, about a

dose of three micrograms per day will give you a one-in-a-thousand cancer risk.

Now, when you think about dislodgeable arsenic per hundred square centimeters, 100 square centimeters happens to be almost exactly the average size of the palm of a single hand of a four-year-old. So I like to think of this data in terms of micrograms on a palm print of a four-year-old.

And you can see, compared to the three-micrograms-per-day dose, the arsenic that could end up on a child's hand, that could rub off on a child's hand from the arsenic-treated woods, is far, far higher than three micrograms. And you can make assumptions about does the wipe take off more arsenic than a hand would, how much of that arsenic from a child's hand would end up in the child's mouth, how much would be dermally absorbed.

But once you get into several hundred micrograms on a child's hand compared to the three micrograms per day at a-one-in-a-thousand cancer risk, it's really pretty easy under anyone's exposure scenarios to get up into the range of really high cancer risks. It's easy to get three micrograms per day exposures for kids. Next slide.

If I could just go back. I'm sorry. I neglected to tell you how we actually used this data in the model.

In the model, each of these vertical lines represents an individual play structure; and a model child is randomly assigned one of these play structures. And then the concentration within that play structure varies randomly each time a child goes out to play on the deck or on the play structure.

The next slide represents studies. These are studies that have measured arsenic levels in the soil beneath arsenic-treated wood. There have been a number of studies done. One, the State of Connecticut measured arsenic levels in soil beneath seven decks, sandy loam soil. And two of those seven studies found quite high concentrations up to 350 milligrams per kilogram.

Now, just to put that in perspective when you're doing like a hazardous waste site cleanup, cleanup levels can be 10 milligrams per kilogram, 20 milligrams per kilogram in that range. So these are pretty far above what would get you into a superfund cleanup level at hazardous waste sites.

Most of the other studies represented here were done in sandy soils. A number of structures were tested in Florida, mostly sand, an Osmose test facility in the State of Florida, and an additional study conducted by the wood industry, SCS 1998. That study focused on 10 prefabricated decks in the State of Virginia.

Now, the residue levels in the soil were lower than what's been found in other studies. The study author over-speculate that maybe that's because sawdust was not generated because the deck was constructed off-site.

In our model, as a child is initiated in a model run, I randomly select one of these distributions to represent the soil beneath that child's deck or that child's play structure. And that represents the soil the child's exposed to.

However, since these data are dominated by these prefabricated decks and prefabricated decks aren't really that common, I only allowed a 10-percent chance that each child will be exposed in a prefabricated deck scenario. And other children are exposed to all the other distributions that represent structures that are constructed on site.

Okay. The next slide represents how much dirt kids eat. And EPA has been around and around on this issue, summarized it in more than one document. These are five of the key studies that are often cited that EPA bases its exposure estimates on.

We've reproduced the data from these studies, based on the distribution statistics. And what we do in the model is randomly assign a child to each one of these measured distributions. This is

how many milligrams of soil per day are ingested incidentally by the child. And then we divide children up into high-, medium-, and low-exposure children to simulate maybe the different ways that children play. Some children just maybe play a little more intensely than others.

Then we randomly select for each play day a soil ingestion value within that third of date, either high, medium, or low. So soil ingestion in our model varies as well. And we have some children who ingest quite a bit of soil just as happens in real life.

Okay. Next, we get to body weight. And this is one of the key differences, also, in our Monte Carlo assessment compared to the point assessment done by some of the Agencies.

We used NHANES data from CDC for 6,000 children to generate body weight distributions that represent the 1st to the 99th percentiles of children through time, from one year of age to seven years of age, our simulation period.

In our model, because Enhanes doesn't measure an individual child through time, we assume that, say, a child who's born at a first-percentile weight stays at a first-percentile weight through seven years of age. So a small child in our model stays small, and a large child stays large.

As each of the one million children are run through our model, we start the simulation by selecting the percentile of body weight that we will use through the child through the model run. And the next thing we do is calculate the body surface area of that child. And, obviously, body surface area is a function of body weight.

And we, again, fall back on NHANES data for body surface area. And we use a regression from Gehan and George, 1970, a study reviewed by EPA, to form this graph that gives surface, the ratio of sufficient area to body weight, on the Y axis as a function of weight in kilograms.

So for each child as the model marches through time we update that child's weight monthly and use this regression curve to calculate a new surface area for that child each month as the model marches on.

So the surface area of the body, of course, is used in dermal absorption pathways. And we've looked at a couple of different scenarios in our model. But our base scenario uses legs, arms, and hands as a possible surfaces that are exposed to soil, that soil would adhere to. And in our model, we assume that only an area equivalent to the palms of the hands, the back of the forearms, or about a quarter of the arms, and the back of the legs or about a quarter of the legs, are exposed to soil.

And our dermal absorption, also, includes dermal absorption of dislodgeable arsenic. So those same surface areas are used for the dislodgeable arsenic dermal absorption pathways.

And that body parts I forgot to mention, the surface area of the hands and the legs and the arms, are based on regressions that are from data presented in EPA's Exposure Factor Handbooks.

Next slide. Okay. So our basic simulation, we do a couple of things. We have the four basic parameters that we allow to vary; and that's body weight, body surface area, dislodgeable arsenic on the wood surface, soil arsenic concentration in the soil beneath the structure, and we, also, allow soil ingestion to vary.

And beyond that, we use parameters that are provided in the EPA document presented to you guys at this meeting. And we compare that to the assumptions that were used in the wood-industry sponsored study done by Gradient this year. And I imagine they'll present some of that data as well at this meeting.

So this is a pretty dense overhead, which I apologize for; but it gives all the details of what goes into the Monte Carlo risk analysis for these different parameters. And if I could just run through them really quickly if you guys can stay awake.

First, dislodgeable arsenic, one of the big assumptions is that

goes into the risk analysis is how many handloads of dislodge arsenic are ingested per day. The Gradient scenario assumes a quarter of two hands, so half of one hand. EPA's assumptions, EPA looks at two scenarios, .8 handloads per day is an average up to 4.95 as a reasonable maximum exposure.

And that's all based on their assessment based on video studies done by EPA and others. And the assumption that the average kind of hand exposure that a child might have is to put about three fingers in their mouth, remove about 50 percent of the dislodgeable arsenic on the hand. And kids, it turns out from these extensively reviewed video studies, put their hands in their mouths about nine and a half times an hour. That's an average up to 20 times per hour. Some studies show much more than that.

In our EWG 2001 as a final column there, that's what we've assumed in our scenario. We just used the average here. We don't try to simulate high exposures. So on every model child, all our one million model children, are nine and a half times an hour they put their hands in their mouths for the hour they're playing on the deck or the play structure.

Then other fixed parameters, bioavailability of ingested dislodgeable arsenic, the wood industry assumes about half. EPA, in

our assessment, we use about 100 percent. The fraction of dislodgeable arsenic that's absorbed through the skin in the version presented to CPSC that was considered negligible in the wood industry study, we've used EPA's assumptions, which is 6.4 percent absorbed.

How much soil children eat. Twenty-five milligrams per day in the Gradient assessment. EPA has proposed 100 milligrams per day as an average from all the available data and a reasonable maximum estimate of 400 milligrams per day. Of course, that's one of our variable parameters; so we let that vary for each child.

Bioavailability of arsenic from ingested soil ran from 16 to 25 percent, depending on which document you read.

How much soil adheres to skin? The wood industry study uses .2 milligrams per square centimeter. EPA has suggested 1.45 milligrams per square centimeter. That comes from a potting soil study but, also, happens to be the average value of what's been measured for wet verses dry soils, how much adheres to children's skin. We've use EPA's parameter in our model.

So then how much of the soil arsenic is absorbed through the skin versus the dislodgeable arsenic? The wood industry study assumes 3 percent. EPA and our assessment are 6.4 percent.

And then the important parameter of what is the level of dislodgeable arsenic, the level on the wood. And the wood-industry study has chosen to use nine values from a single industry sponsored study. The maximum there is 13 micrograms per 100 square centimeters.

That's really low compared to many of the values that have been measured. We found pretty commonly hundreds of micrograms per child's handprint, 100 square centimeters were dislodgeable.

EPA, of course, is pending on that decision. They have a proposed sampling plan out to deal with that.

Dislodgeable arsenic on wood. We've based our analysis on the 19 distributions that I showed you previously. These are measured distributions on individual structures. The range goes from about zero to over a thousand micrograms per hundred square centimeters for a particular new wood sample that was unsealed.

Next, what has been assumed through these studies for the arsenic level in the soil industry sponsored study was used, SCS 2000, reasonable maximum of 30 milligrams per kilogram. EPA is pending on that one again. Proposed a sampling program to better define soil concentrations.

We've used the data that exists for 27 individuals structures and

the soil concentrations beneath the structures. It's quite a lot of data. It ranges from nondetectable to levels to 350 milligrams per kilogram in the soil.

This is a really important parameter, the next one, the wipe-to-hand transfer coefficient. So the question is: If you get a certain amount of arsenic on a wipe sample, how does that compared to what would be on a hand sample if a hand had swiped that same area?

The wood-industry study gets around that by using a single study that directly measured hand data. EPA is proposing that they'll assume that what gets on the wipe is the same as what would get on a hand.

We've sort of gone two ways on that. We first present data assuming that about a quarter of the wipe arsenic would end up on the hand. And then we've said, well, what if instead pretty similar what ends up on a wipe is also the same as what ends up on the hand. And we present both of those scenarios here.

And one quarter factor, or it's actually 4.6 times as much on a wipe as is on the hand, has a basis in an industry study conducted by SCS in year 2000, I believe, that compared -- they took the same samples of wood and used wipes, dry wipes, on that wood and also

hand wipes. And if you compared those samples directly, the data is extraordinarily variable. But the median -- but sometimes the hand concentrations are higher than the wipe concentration and sometimes lower.

But the median value is 4.6 which is what I've used in this assessment. So four times six times the arsenic is 4.6 times higher on the wipe then on a hand given that you're swiping the same area.

Next is body weight. Gradient and EPA assumed fixed body weight for the age group that they look at. And we, of course, let the body weight vary. Each child is given a percentile body weight from the 1st to the 99th percentile based on N//HANES distributions.

And last but not least is the body surface area which goes into how important dermal absorption is.

Gradient assumes entire legs, arms, and hands for soil; nothing for dislodgeable arsenic. EPA, it looks like, is proposing the entire surface of arms, legs, and hands. We've assumed partial arms, legs, and hands: a quarter of the legs, a quarter of the arms, and the palm area of the hands.

So now we get to the actual computation which is excess lifetime cancer risk. This particular graph that I'll show you is based on just the Ira straight linear 1.5 milligram per kilogram day inverse

of that slope factor not the NRC's new recommended values which look like they're higher.

We simulated, first, a group of children who are exposed to a play structure three times a week for an hour each time. And we assumed that this represents about a third of all kids get this fairly regular play and that turns out to be 10 million kids out of the 30 million children in the age group that we're simulating.

So you'll see the 10 million kids on the X axis here. And the conversion of our chart from the PC to the MAC messed up our title on the Y axis, but that's Excess Lifetime Cancer Risk on the Y axis.

So for the baseline assessment, if you could pull that curve up. This line is only ingestion of dislodgeable arsenic on play structures for kids who play three hours a week on these structures. You can see even in this baseline assessment, if you follow over 10-to-the-minus-4 line, you have about 15 percent of all our kids are above 10-to-the-minus-4 risk level. So we're already in the extremely high zone for the single exposure route and exposure pathway for a good number of the kids that we're looking at.

Now as you add routes and pathways on top of that, which is what reality is, the second line adds to that graph dermal absorption from the dislodgeable arsenic. So if a quarter of the legs, a quarter of

the hands, and the palms are exposed to dislodgeable arsenic, 6.4 percent of that dislodgeable arsenic absorbs through the skin, you get this additional risk.

Next, we asked the question, well, what happens if this child is also playing in contaminated soil under this structure. So the third line represents soil exposures. And that's assuming that there is soil ingestion each day that comes from soil that's been contaminated with arsenic.

We use measured distributions for soil ingestion. And it's just more risk and more risk piling up as you add these pathways. And these are, of course, real pathways that many kids are exposed to. So then this is kids three times a week.

We then ask the question, well, what happens if a child is going to a school or has a play structure at home? So they're regularly playing on pressure-treated wood and they have a deck on the deck of their house; and they play on the deck, or they store toys under their deck; and they're exposed to arsenic on their toys that are on the deck itself.

Well, for those kids, the risks get even higher. And these children are assumed to -- that top line represents children three hours a week on a play structure and three hours a week on a deck. And in

the dislodgeable arsenic residues in the model, we don't distinguish between play structures and decks because the wood is all the same.

So if a study happened to look at a play structure, we'll use that distribution in the model, also, to represent levels that would apply to a home deck.

And you can see from this top risk curve, we've got 60 percent of our children exceeding a 10-to-the-minus-4 risk. In this scenario, we assume as a baseline 10 million kids are getting these exposures. So the risks are just extraordinarily high.

And in these exposure parameters, you know, we did our very best to pick reasonable estimates and, in some cases, probably tend to underestimate the exposures. So that's our baseline scenario.

For the parameters that are fixed in the model, we have basically used EPA's assumptions that they've proposed for your meeting this week. We also did a comparison analysis for the wood-industry study that was presented at the last CPSC meeting.

The top line is what we call the "EPA scenario." It's basically EPAs assumptions with our variable parameters on top of it. So variable body weight, variable dislodgeable arsenic soil and soil ingestion.

Then we said, let's use those same variables. But for the fixed

parameters, let's use the assumptions that were used by the Gradient study. You still get 60 percent of all kids. And under the wood-industry assumptions exceeding a 10-to-the-minus-5 risk. So it's really difficult to get under anybody's scenarios under a one-in-a-million risk for children who are playing pretty regularly on this wood.

Now, let's look at what happens. You know, our baseline assumptions are that only a quarter of the dislodgeable arsenic would end up on a child's hand, about a quarter. So let's look at what happens if, as EPA's proposed, all of that dislodgeable arsenic ends up on the children's hands.

This, again, is our baseline scenario where a quarter of the dislodgeable arsenic is allowed on the skin. If you instead assume that there's a one-to-one transfer ratio between the wipe studies and the hand studies, you, of course, jack up the risks by that much more.

We have, in this case, two million children; 20 percent of the children in our model exceeding a one-in-a-thousand cancer risk under a one-to-one transfer assumption. These models get pretty high pretty quickly under different assumptions.

Now in the last graph, we've done an assessment of, you know, what happens if instead of using the IRIS default slope factor of one-

and-a-half milligrams per kilogram day to the minus 1, what happens if you instead use some of the LED_{01} values computed in the NRC update.

So what I've done is just to take some central tendency estimates from the NRC report for both bladder cancer and lung cancer from the Taiwanese studies; and I converted that to a daily dose, assuming 70 kilogram body weight and a liter per day water ingestion.

I computed the same scenario using instead that cancer potency factor. And you can see that our baseline scenario is on the bottom, and that's the bladder cancer risk using the IRIS one-and-a-half default linear slope factor.

The line on top of that is what happens instead if you use the NRC LED_{01} with the assumption that the slope is linear. And we extrapolated linearly from the 1 percent. And then, of course, that 2.8 factor gives you elevated risks compared to the IRIS factor.

Then we looked on top of that. What happens if you look at lung cancer? The LED_{01} sort of a central tendency from the NRC report with the linear extrapolation, the risks are even higher.

And then as the final curve, which I guess we lost, we added bladder and lung cancer, which is presented on your overhead. And

the sum of those two cancers is based on the NRC central tendency estimates for the LEDO1s.

So just to sort of wrap this up, what I'd like to leave you with is there are a lot of studies out there already on dislodgeable arsenic levels in existing structures. There are 19 good studies that can already be used. There are lots of studies out there on soil arsenic levels beneath the structures. We simulated 27 of these structures from existing studies. And EPA has compiled much more data then we were able to compile. So they have even more than this.

So the point I'd like to leave you with, one, is that there are data already out there that are perfectly sufficient to do a risk assessment that shows extraordinarily high risks for some of these kids. And our data are dominated -- the soil data is dominated by sand, so the arsenic concentrations are biased low. The dislodgeable arsenic concentrations are dominated by sealed structures. So, again, the dislodgeable arsenic concentrations are biased low.

So any additional studies that go out and sample more and more and more structures will take another year and will probably make these risks look even worse. So that's one point I'd like to leave you with.

And the final point I'd like to leave you with is that these risks

for many, many kids, 15, 20 percent of the kids, are really pretty extraordinarily high. And on top of that, they're drinking arsenic-contaminated water; and we didn't incorporate those risks in our analysis.

DR. ROBERTS: Thank you for your presentation. Are there questions from members of the Panel?

MS. HOULIHAN: I'm glad I was so clear.

DR. ROBERTS: Dr. Thrall.

DR. THRALL: I just had a question for clarification. I'm not familiar with this. Is the amount of soil ingested actually measured in some way, or is that just derived from the hand-to-mouth contact in the amount of soil on the hands?

MS. HOULIHAN: The ingestion of the dermal absorption arsenic -- I mean the dislodgeable arsenic -- is based on the hand-to-mouth transfer coefficient. The ingestion of soil arsenic is based on these key studies that have measured childrens's exposure to soil mainly through trying to recreate soil ingestion through measuring body fluids and arsenic that's excreted or, you know, soil contaminants that are excreted.

DR. THRALL: So they're actually measured then.

MS. HOULIHAN: Right. In the five key studies that we've

used, there are measurements for each point on that chart. That's in your presentation materials. That represents one child's daily ingestion of soil that was computed in these studies.

DR. THRALL: Okay.

MS. HOULIHAN: These are, also, the methods that EPA has put forward as how they propose to look at ingested arsenic for the two different possible pathways.

DR. ROBERTS: Dr. Kosnett, then Dr. Ginsberg.

DR. KOSNETT: Did you allow certain variables to vary independently, for example, the amount of dislodgeable arsenic and the amount of arsenic in soil below the structure; or did you somehow tie those together?

MS. HOULIHAN: They weren't tied together. They were independent. So a given structure -- and I think in real life, they'd probably find that they're independent because the arsenic level on a structure will depend so much on the age of the structure and the condition of the wood; and the arsenic level in the soil depends really strongly on how often the wood might have been sealed and the soil type and the conditions, the weather conditions. So those are just going to be all over the board.

DR. KOSNETT: Is the lack of independence borne out by

empiric studies, do you know?

MS. HOULIHAN: I haven't seen studies that have tried to address that question.

DR. ROBERTS: Dr. Ginsberg.

DR. GINSBERG: I'm curious why you didn't run any of your Monte Carlo simulations based upon -- and maybe you did. It just wasn't clear from your presentation. Based upon your figure where you had the hand-rub information. I mean, you're doing this extrapolation from the swipe to how much gets on the hand.

MS. HOULIHAN: Right.

DR. GINSBERG: But we have data on people rubbing decks with hands.

MS. HOULIHAN: And what we did was -- and I didn't mention this -- use directly that hand data. So the hand studies, each of those hand studies, represents a structure. And we use that data directly. But then if we chose a structure where only wipe data was available, then depending on the scenario, we either adjusted that or we did a one-to-one transfer coefficient.

DR. GINSBERG: So that's part of your distribution for the dislodgeable data set there.

MS. HOULIHAN: Right.

DR. GINSBERG: You said there's 19 structures. So those three data sets are part of that 19; is that what you're saying?

MS. HOULIHAN: I think, actually, the 19 does not include the three hand structures; but I'd have to go back and check. Sean? It does include. Nineteen does includes the three hand studies. Sorry.

DR. GINSBERG: It's a little hard to see how you exactly put together these distributions and selected points off of them for your high-, intermediate-, and low-exposed groups.

MS. HOULIHAN: That's for soil ingestion only; right?

DR. GINSBERG: So for the dislodgeable --

MS. HOULIHAN: Dislodgeable --

DR. GINSBERG: Why don't you describe how you compiled all this data and picked points off?

MS. HOULIHAN: So a child is introduced into the model. A body weight percentile is selected. So that kid is, say, a 27-percentile weight kid, which is maintained throughout the seven years, the six years of the model. And then the child, in the beginning of the model, is assigned one of these dislodgeable arsenic profiles from an individual structure. And they're also assigned one of the soil arsenic distributions. And those are selected independently.

A child is given a 1-and-15 chance of getting one of the samples

of new wood from our data, which our samples are quite high. I didn't mention this detail. But in those really high concentrations, we only let persist for one month and then we assumed there is some process where that arsenic is washed off the wood.

The aged structures, we let those structures, that whole distribution of dislodgeable arsenic, persist through the child's simulation period. Just one structure. Yeah. So that persists.

And then we give a child -- this is a level of detail you might not want to know -- a one-in-four chance of moving every year because children move. And if the child is selected to move in the model, we pick a new structure and a new soil distribution.

So the child is marched through time in this model. Three times a week, three hours a week, they're exposed to a deck and play structure, depending on a scenario. And then monthly, the body weight is updated and the surface area of the child is updated. So then we just continue to compute this average daily dose through time and in the end divide by the lifetime of the child.

DR. ROBERTS: Let me ask: Is there a written description that has these methodologic details that could be available for the Panel?

MS. HOULIHAN: I did write up a methodology that you should have a copy of.

DR. ROBERTS: We do have this, but I haven't had a chance to read it. It just appeared over lunch.

MS. HOULIHAN: Yeah, you only just got it.

DR. ROBERTS: Will there be information in here that will answer Dr. Ginsberg's, and perhaps others, question about this?

MS. HOULIHAN: At the level that I -- yes, the descriptive level that I'm answering them now and in combination with the data graphs that I've presented here is a pretty good summary of everything we've done in our method.

DR. ROBERTS: Okay. Thank you very much. Dr. Mushak, you had a question and then Dr. Smith.

DR. MUSHAK: Yeah. A comment about and a question about the role of direct oral contact by kids.

We know that kids chew on surfaces. We know that with certain toxicants they can be severely injured as in the case of lead paint chewing. Now, neither you nor the scenarios proposed with OPP try to get a handle on that. And I find that a big gap. Direct oral contact cuts out the pathway middleman of the hand contact so that whatever sequences of uncertainties that you have with direct oral contact, at least it's recaptured by having to avoid all of these parameters that go into a hand transfer and efficiency of removal, et

cetera.

I think that's a type of exposure route that has to be developed by the Agency.

MS. HOULIHAN: That's a great suggestion. I didn't have data available to include that. But, obviously, there are lots of kids who mouth the wood and their exposures are going to be even higher.

DR. ROBERTS: Dr. Smith.

DR. SMITH: Two questions, if I may. The first one is a follow-up on Dr. Ginsberg's question. And let me see if I can ask the same question a slightly different way.

When I'm looking at your distribution, say, for dislodgeable arsenic from the wipe, so you have the various spreads of the data. How is it that you're actually parameterizing statistical distributions for use in the Monte Carlo? Are you just resampling from these data? Are you using the data to fit an empirical distribution and then you're putting bounds on percentiles that you can sample, or are you fitting like a log normal or normal? So if you could just tell us how you're handling and that and how you're handling the extremes.

MS. HOULIHAN: Some of these data we have the data directly for, and others of these distributions, we have the statistics for our mean and a standard deviation and sometimes a range of measured

values. And in those cases when we have the statistics, we generate a distribution that fits those statistics and force the min and max to conform to the measured minimum and maximum values.

DR. SMITH: So the max and mins make the boundaries on what you're going to sample.

MS. HOULIHAN: Right, right. And in a few cases, we don't have those values; and we just let the model generate a data set that fits the other statistics.

DR. SMITH: And one more question, if I may.

I'm struck in looking at this, which is a rather nice way to present the data, that the variation within a site is just as large as the variation between sites. It looks like it's a little over an order of magnitude for any specific structure and the same between.

I'm curious as to what you think it is that you're modeling with this sort of characterization of variability, whether you think it's variability or uncertainty in the measurements or exactly what's going on here. And I ask that in part because I notice that you're using some of the data we generated. And as you know, from the work we generated, yes, there is a number of different individual observations; but those observations, you know, the focus of our study, was understanding the phenomenon not trying to get data.

MS. HOULIHAN: Right.

DR. SMITH: So each of our data points normally reflect different lengths of wood that the hand's been rubbed on for different durations of time, lots of variations like that. So I'm curious as to what your thinking is, what you're characterizing here.

MS. HOULIHAN: I'm an engineer, and engineers tend to like things standardized. But in this case, I think that the huge variety of wipe methods and contact methods that have been used are really valuable in simulating the different -- and kind of getting at the question of kids play on this wood in all different ways.

Some kids will pretty aggressively be rubbing the wood and other kinds will be lightly touching the wood. And I think some of the differences of wipe methods and hand-study methods can get at some of that variability. And in this case, you really might not want one single wipe method that does things one certain way.

DR. SMITH: So if I understand your response, your view is what you're getting at is variability in potential loading onto a hand across structures and within a structure.

MS. HOULIHAN: Yeah, for the different ways that kids play on this wood.

DR. ROBERTS: Okay. Any other questions? Dr. Kissel.

DR. KISSEL: You've projected doses and then taken that to carcinogenic risk. You could also project what you ought to see in kids's urine if they had doses this high. Have you done that? Have you looked at those numbers and then compared that result to what actually shows up in kids for kind of a check on the general validity or likelihood that your numbers are good?

MS. HOULIHAN: That's a great idea. If there's a large scale study of arsenic levels in children's urine as a reality check for how many kids get these really high exposures.

But, you know, my sense is that -- I know. I have two kids. One's two and one's four. And they are regularly playing on pressure-treated wood. And I think a lot of people who have kids, once you start working on this issue and thinking about it, you realize how ubiquitous the wood is in our lives. It's just in every park you go to, everybody's backyard. It's everywhere.

DR. KISSEL: Well, there are studies out there not oriented toward this but because of other types of arsenic contamination. And these things have been out there a lot. And if there are a lot of people exposed, then you should see some of these kids show up in those other populations. And it's kind of an obvious thing to look for.

MS. HOULIHAN: Right.

DR. ROBERTS: Dr. Wargo. Thank you for flagging.

DR. WARGO: I also have an interest in the methodology that you used, and I assume that you'll provide material that will clarify that.

But I guess the basic question is: Are you adding up the exposures from the different sources for each individual child, or are you changing both the source of the exposure and the child as you accumulate the exposure?

MS. HOULIHAN: Each child is preserved. The risk for that individual child is preserved throughout the model for each of the eight possible combinations of pathways and routes.

DR. WARGO: And do you carry that child across time as well?

MS. HOULIHAN: Yes. We carry each child from one year of age through seven years through the model and maintain a kind of running average daily dose.

DR. WARGO: Have you done any studies --

MS. HOULIHAN: Through six until their seventh birthday.

DR. WARGO: Sure. Have you done any studies looking at the high-end exposures, the group that is appearing at the upper end of your curve, to understand what factors might be driving that? I mean, if you look at the two-year-olds or if you look at the six-year-olds that

spend an inordinately high amount of time in the playground or on certain sets of certain ages, I mean, what factors do you think, after doing this, are the ones that are really driving the high-end exposures?

MS. HOULIHAN: Well, the most important exposure pathway seems to be ingestion of dislodgeable arsenic. Because in general, the data we have on soil, the arsenic concentrations are fairly low. So that's the important pathway.

Now, we didn't break down the high-exposure kids to figure out what we could. But, you know, who is that kid and what's their body weight and what structure they're playing on. But you would obviously guess it's the small kids playing on high arsenic structures that that combination will automatically get you up into the higher range.

You know, I don't know the relative importance of all the other factors. But you know, all our kids in this model play for a set period of time. So it's not the time that they're on the structure. They're all three hours a week.

DR. WARGO: Just one final question. Has the Agency reviewed their methods yet?

MS. HOULIHAN: Well, they've reviewed plenty of our Monte Carlo risk assessments but not this particular one.

DR. WARGO: Okay. Thank you.

DR. ROBERTS: Dr. Thrall.

DR. THRALL: Just one more question. Regarding the surface area of the palm, 100 centimeter square is one palm of what age child?

MS. HOULIHAN: It's a four-year-old's palm, one palm. It's a single palm area of a four-year-old.

DR. ROBERTS: Dr. Bruckner.

DR. BRUCKNER: Hi. Jim Bruckner. Just a question. I'm not sure if you addressed this. Have you determined if the plus III or plus V arsenic that's coming off on the hand is being ingested?

MS. HOULIHAN: Oh, a number of studies have addressed that. I think they're summarized in the EPA document. But I didn't address that directly in our assessment.

DR. BRUCKNER: Can I ask someone in EPA? Has that been determined or established?

THE EPA: We don't feel that there are adequate data to really determine the species, so we're assuming total arsenic.

DR. BRUCKNER: Okay. What I heard, I think was, that it's plus III that goes into the wood.

THE EPA: Well, for the chromium, it's plus 6 and then it converts to plus III.

DR. BRUCKNER: I'm sorry. Arsenic.

THE EPA: Pardon?

DR. BRUCKNER: Which form of arsenic goes in?

THE EPA: Well, in the formulation, it's pentoxide. I'm not sure about the fixation.

DR. BRUCKNER: I'm just wondering if this is the major route of exposure. Just if anyone has any idea of what form?

DR. MUSHAK: I think in quick response to your question At the low intake levels, we're talking about, I think there's a vast amount of data over the last 20 years that show that the arsenic III and arsenic V are interchangeable in toxicity. That notion that V is less toxic than III springs from early data on acute exposures of animals.

I think that all of the biotransformation data of Marie Vahter and others, and Vash Aposhian, who's in the audience, show that small amounts of pentavalent immediately transformed to trivalent.

So if your question is geared to relative toxicological potency, then there's no difference.

DR. ROBERTS: Okay. I think --

DR. BRUCKNER: I was thinking more about kinetics absorption.

DR. MUSHAK: Well, I think the same would apply. I seen no

data on low amounts of mobilizable arsenic at these microgram levels that would suggest that.

DR. ROBERTS: We kind of need to move along in the comments. Dr. Styblo, I believe you have a question; and then we can --

DR. STYBLO: Just a very short comment. When we are talking about arsenic V and III, obviously everyone means inorganic. We still don't know how much organic methylated arsenic plus III and plus V is present in soil and on the surface of the wood. So all your data are based on the risk assessment after ingestion of inorganic arsenic. If there is any other species, all these numbers would look different. In fact, we don't have data which could calculate those numbers at the moment.

DR. ROBERTS: Well, your presentation has obviously simulated a lot of interest and discussion among the Panel. Thank you very much. I appreciate it.

MS. HOULIHAN: Thanks.

DR. ROBERTS: Our next commentor is Chris Williams, who will followed by Ligia Mora-Applegate.

DR. WILLIAMS: My name is Chris Williams. I am an environmental toxicologist from Ecology and Environment, an

environmental consulting firm based in Buffalo, New York. I'm actually in Tallahassee, Florida.

I have -- it's really a question. It's not a comment. And I can probably just as easily make it and then sit back down. This kind of addresses some of the information that was presented to us concerning hazard endpoints this morning by Dr. McMahon, and it kind of gets to the issue of maybe doing a reality check on all the science that we're talking about.

I'll pose the question formally. And if I need to repose it in more general terms, I can do so.

My question is: How will the available body of human literature concerning exposure to CCA-treated wood and/or residue-containing soil be used to assess hazards and risks in children?

Now, if no such literature exists or if the literature indicate a general lack of hazard or risk or perhaps at best diminutus hazard or risk, how do those concerned with making these decisions propose to address this in the risk assessment? And if it's a data gap, how would that be proposed to be addressed?

DR. ROBERTS: Okay, well, I don't know that there's anyone on the Panel who can answer the question for you. Is there? And I guess it's really a question of are you posing that to the Agency, or is this

part of your comments to the Panel?

DR. WILLIAMS: To the Panel.

DR. ROBERTS: I don't know. You are really asking how the Agency is going to respond to the outcome of an assessment. And I don't know that the Panel --

DR. WILLIAMS: Well, I guess what I'm getting at, Steve, is are there other data out there, other than data concerning drinking water exposures and those sorts of things, that might more closely mimic the type of exposure that we're talking about here and give us a feel for what the effects are, what the risks are, that sort of thing. I think that's where I'm coming from.

DR. ROBERTS: The Panel's more used to asking questions than answering them from the commentors, I have to tell you that. I don't know if anyone on the Panel wants to take a shot at that or not.

DR. WILLIAMS: I guess a traditional way under risk assessment is to consider it as an uncertainty in an uncertainty section. But I guess in this instance, if there's some way that it can be considered. That's all.

DR. ROBERTS: Thanks, Dr. Williams. Next commentor is Ligia Mora-Applegate, who will be followed by Pascal Kamdem.

MS. MORA-APPLEGATE: Good afternoon, Mr. Chairman,

members of the Panel, EPA. I'm really pleased to be here, and I'm grateful for you to listen to me.

OPP has proposed several exposure assumptions to be used in the evaluation of potential health risks to children in play structures made with wood treated with chromium copper arsenate. While this assumptions may be used to represent an average situation for the whole nation, there is concern that they might underestimate exposures occurring in the State of Florida. As you guys know, Florida has a wonderful climate, especially in the winter. That is a hint to come down to see us.

But anyway, in particular, there are some indications that the proposed exposure frequently of 130 days a year may be too low given there is some indication that the proposals given that the assessment will focus on one-to-six-year-old children.

A large proportion of these populations attends day care facilities that operate most of the year. I would say 250 days a year. And we may equate that if you imagine the standard number of days assumed of work by the parents. And given the variable weather condition that pervades in Florida, a reasonable maximal exposure to playground equipment likely equates with the number of days per year children attend day care facilities.

A related issue is that OPP has proposed one hour a day and three hours a day as central tendency and reasonable maximal exposure for playing in play structures made out of CCA-treated wood.

There are more in Florida. Cold weather is sporadic and rather rarely persists throughout the day. Also, rain events are usually short and occur in the late afternoons or evenings like rain showers. These weather conditions, not common elsewhere in the country, point to the fact that Florida may harbor conditions reflecting reasonable maximal exposures.

The issue that should be considered relates to the fact that CCA-treated wood is increasing ubiquitous especially in states such as Florida where wood-destroying organisms are a major problem.

The statistics show that the amount of CCA-treated wood is increasing exponentially in Florida. Children are likely to be exposed to CCA-treated wood not only on the day care facilities or public playgrounds but also in their homes. These other sources of exposures should be formally addressed through a comprehensive risk assessment.

DR. ROBERTS: Thank you. Are there any questions for Ms. Mora-Applegate? Well, one. Don't run away.

DR. GORDON: Are there any statistics on the percentage of play structures or decks that is greater in Florida or Hawaii than other states?

MS. MORA-APPLEGATE: I don't know about that specifically. But I can tell you that the numbers of days that children do play is greater.

DR. ROBERTS: Any other questions? Dr. Smith.

DR. SMITH: I am wondering if I am asking the same question as Dr. Gordon just asked.

Again, the Agency is relying on their Exposure Factor Handbook to give data. For example, from survey data for the amount of time a child would typically spend outdoor on a certain type of structure like Playscape, is my recollection.

And so I think one of the questions we're asking you is: Do you have any Florida-specific data that would suggest that that really is an underestimate for your particular location, either in terms of days per year or in terms of hours per day?

MS. MORA-APPLEGATE: We called a few of the day care facilities that our children do attend, and that's what they told us, that they do play just about every day that they are there and also three to four hours a day. But it is not a formal study. It's just a few phone

calls.

DR. ROBERTS: Other questions? No. Thank you very much.

Our next commenter is Pascal Kamdem, who will be followed by Dr. Vashen Aposhian.

DR. KAMDEM: Good afternoon, Chairman and members of the Panel.

I would like to share with you today the result of the work that we did on chemical analysis of the dislodgeable compound from the top surface of CCA-treated wood. I'm an Associate Professor at Michigan State University. I've been working with wood preservatives for the last 10 years. Next please.

The objective of this work, again, is to characterize the dislodgeable compounds on the top surface. Again, this is the top surface of CCA-treated southern pine planks that was used to construct and build a deck.

We received the sample. The sample was shipped to me from Fayetteville, Georgia. And those samples were used in the construction in a deck for about 16 months; and the species, of course, was Southern Europe pine.

The size of the sample was one-inch thick by five and three-eighth inch wide and 24 inches long. And the chemical that was

used for the pressure treatment was CCA-type C. That means we have about 17-percent copper, 44.5-percent chromium, and 30-percent arsenic. And it was treated by the company named Treated West Southern (Ph). And their attention on the sample that we received, the wood sample that we received, and that was exposed for 16 months was about 0.37 pound per cubic of total oxide using the density of 32 pound per cubic foot.

And you can see the different concentration in term of elemental arsenic, copper, and chromium; and the next column is the oxide. We just multiplied the elemental by a factor to get the oxide. And we obtain a ratio. The ratio between chromium arsenic copper in elemental again is 55 -- 51 to 35 to 14. Next please.

These are the protocols that we used to obtain the difference solutions, solids, and for analysis. First, the wood plank was analyzed for copper chromium arsenic and for chromium 6. And then we washed the wood plank surface with water and also by brushing with a test tube brush about five times. This is to simulate the worse scenario.

And then the solution that was obtained contained water and some wood residues and sand. And we feel using a glass wool to remove that the particle that was higher than 0.2 millimeter. And

from the liquid, we did some rotoevaporation at a temperature lower, I would say, than 60-degrees Celsius. Because if you want to get a concentrate that have about 1 milligram of arsenic per mil. So we went from about 5,000 mil. that we used to wash 86 planks of wood down to 10 mil.

Of course, from the 5 mil. that was used for the washing, we collected only 3,008 mil. That means 3.8 litre because we got some absorption by the wood during the washing. Next.

So you can see the plank that we received, washing, collecting the dislodgeable compound on the surface. And then you can also see how we used glass wool with the small particles that were removed. And then on the small -- that's the solution that we got, the 10 mil. solution that we obtained. You can see that we have some precipitate after the rotoevaporation. Next.

So for analysis, what we did, we used several techniques. First, we used solid state method because we want to get information on the wood surface itself. Not in water, but just the wood surface as it is. So we use ESCMEDXA, which is an environmental scanning electronmicroscope which is coupled with energy dispersive X-ray, to get information about the atomic composition on the wood surface.

And then after that, also, we used XPS and XRD. XPS stands

for X-Ray photoelectron spectroscopy. It will give information about the surface composition and also some valence state of the different atom that you have on the surface using chemical shift.

And XRD, it's X-Ray defraction. It will give you information about the crystal nature or the amorphous nature of any solids.

And then for the liquid analysis, we used ICP, or an atomic absorption spectroscopy, to determine the amount of copper chromium arsenate, the total copper chromium arsenate.

And then UVVIS was also used to again evaluate and determine the amount of chromium 6 that we have in solution. Next.

This is a summary of the results that we obtained. You can see from the table in red we have the concentration of the different element on the wood across -- this is not just the AWPA assay. This is just across the wood. We got some copper chromium arsenate, but the chromium 6 was not detectable. And the method that we used for that detection limit is about 1 ppm.

And then for the second solid that was removed during the filtration using glass wool, we obtain about 0.3 gram of that solid. This is on oven-dry base. And in that solid, we have copper chromium arsenate which is very low, 0.06, 0.03, and 0.2. And, again, in those solids, there were almost, I would say, wood residues. The chromium

6 was also not detectable.

And then after that, the solid that we obtained in the -- and after evaporation was about 2 gram. Again, you have to remember that is from 86 pieces of wood and the total surface is about 73,100 centimeters square, equivalent to a deck that will measure about 8 foot by 10 foot.

So from the solid that we obtained from that, we can see that, yes, we have some copper chromium arsenic. Again, this is elemental elements. And then the chromium 6 was not detectable. But in solution, we detect, we have some copper chromium arsenic and a little bit, just a very little bit of chromium 6, about 0.003 percent was chromium 6, that we got after evaporating from 3.8 litre to 10 mil.

Therefore, I would say that, yes, we have some chromium. The total chromium it's about 2001.6 milligram total arsenic, about 18.8 milligram.

Now, based on this, we want to soluble was this solid. Because after rotoevaporation, we obtained some precipitate so we want to know the solubility.

So we did a quick experiment by just taking the 0.05 gram of the dislodgeable solid, that precipitate after rotoevaporation, and mix again in 100 mil. of the I water. After one hour, we find only 0.02

milligram, about 2-percent copper, 1-percent chromium. This is again total. And arsenic was not detectable at that level.

And then we went and we continued the mixture for about 24 hours. And we increase the amount of copper from 2 to 4 percent and also doubled the amount of chromium, total chromium. And, again, we didn't detect any arsenic, but we continued to do some analysis. We sent a sample for ICP analysis because of the detection limit of arsenic using AASR or ICP is different. Next.

So this is a micrograph of the solid after rotoevaporation. And you can see the average particle size here is around 100 micron because this is a 2,500 magnification.

And for the XPS, again, I just want to show you something. I don't have my pointer. Anyway, you can see on the left-hand corner this -- thank you. This is the XPS. For the XPS, first, we survey; and then we got information from zero to, I would say, 1,000 kilo electronvolt. And we went here. We saw some chromium.

You say, oh, yes, since there is chromium, we're going to go again and conduct an experiment for a little bit of a long time to see what kind of chromium it is.

And it's well known from the literature that if you have chromium, the electron 2P one-half and 3P one-half, with the 9.8

electron volt, yes, we know that this is chromium 3. So this study clearly showed that, yes, we have a chromium 3 on the wood surface. Again, this is on the wood surface.

And then the same thing here for the arsenic. This is the specter of arsenic that we obtained. So just in that it's arsenic 5. And, of course, just for oxygen and carbon that we use for the calibration. That's very common on XPS. Next, please.

Now, for the XRD, what we did, also, we cut a little piece of wood on just the surface. And then we exposed that, we ran some XRD to obtain a spectra. And you can see that this is typical of cellulose. This is CCA-treated Southern Europe pine. You can see that that is cellulose; it's well known.

And we do the same thing just by taking copperoxide, arsenicpentoxide, and some chromium and mix together. Just physical mixture is not the same pick any more. You can still see the cellulose here, but you got some defraction angle there because you have some crystal in here.

So suggesting that the solid that we saw on the surface of CCA-treated wood, it is not the same solid that we have. It's not -- I'm sorry. It's not crystal form; it's amorphous.

And then if you go down here. This is the solid that we

obtained by just taking the treated solution and rotoevaporate and then leave it in the lab to dry. And we run the XRD. You can see here it is completely different, have nothing to do with the CCA-treated wood. This is the treating solution here.

But if you take the dislodgeable compound and rotoevaporate and then run your XRD, you can still see your cellulose here and there's a defraction angle here. And I don't know exactly what it is, but it's different what you have here when you fix the initial chemical that you will use for the treatment.

So this study clearly suggests to me that when you treat wood with CCA, number one, you cannot assume that is the same form of arsenic and chromium as you have in the initial treating solution. There is some chemical reaction for fixation going on. So more likely, you have formation of copper chromium arsenic complex. And this has been proven in the literature. But, yes, you have formation of CR chromium arsenic and also copper arsenic. Next, please.

So in conclusion, again, I would say that we have formation of a completion containing chromium, arsenic, oxygen, copper, which is not similar to what we have in the treating solution. And, also, the solid present on the surface of CCA-treated wood are amorphous. There is no crystal. The same type of crystal that we have in the

treating solution is not there. And, also, we have a very low solubility of the amorphous solid that are on the surface CCA-treated wood.

Thank you.

DR. ROBERTS: Thank you for your presentation, Dr. Kamdem. It looks like a couple of questions. Dr. Chou, Dr. Mushak, Dr. Solo-Gabriele, and Dr. Styblo.

DR. CHOU: I have several questions. Probably they're all short. Why do you use 18- or 16-month-old wood? That's the first one. And do you believe that's the most representative of the real world, or whether you have done other wood with different ages?

Also, you described this as a worst-scenario test, that you use water to brush the surface. I wonder what is the pH of the water you use; and, also, how does it compare with the pH of sweat and rainfall. Because just short, sweat is known to extract CCA elements more than water.

And the third one is after 18 months the wood is outside, wouldn't you say that the liftable elements are already gone. So what you would get is those that would stay there. They are not dislodgeable ones, a portion. Well, I think I'll stop here.

DR. ROBERTS: I think I counted four, Dr. Kamdem.

DR. MUSHAK: I'll keep mine to two.

DR. ROBERTS: Wait, wait, wait. Let's let Dr. Kamdem respond.

DR. CHOU: I want my answers.

DR. KAMDEM: Do you want me to respond first?

DR. ROBERTS: Yeah. Rather than pile them on, why don't you go ahead and take the first four.

DR. KAMDEM: We were looking for -- I was looking for a deck that was newly put without any coating, any sealing, because today you have a lot of different formulation of CCA-treating solution and including some water repellants. So we were looking for a deck that was built and without using any water repellants in the treating solution. So that's why we went with the 16-month old deck. And also because Osmose in Buffalo was able to provide us with a 16-month-old deck that was in a house without any occupation, anybody living in the house. That's the answer for my first question.

Now, the next was what was the pH? The pH of the water was 6.2.

DR. CHOU: And do you know - since you described this as the worse-case scenario, how would you compare that with the pH of sweat or in rainfall?

DR. KAMDEM: Well, it's known, it's established, that if you

use a low pH like something with, I would say, oxalate, citric acid, to wash CCA-treated wood, you will remove a lot of CCA. Yes, the pH is very important. But in this study, I did not vary the pH. I just use the pH of the DI water to wash by brushing five times. That's why I say the worse scenario. Brushing five times and then using 60 mil per 860 centimeter square. So that's why I said that it was the worst scenario.

DR. CHOU: I had one more question. After the 16 month, the elements on the outside portion of the wood is probably leached already, and all you're brushing is the outside surface. But in a real playground, children actually rub the wood off and the inner portion would be continued to be exposed; wouldn't that be true?

DR. KAMDEM: Yes, that's exactly true. And my intention is to try to locate a new deck and do a time study on that to see what will be the effect of the time and that propose that the effect also may be the pH of the water. You still have some acid rain. So what will be the factor -- the effect of this on the dislodgeable compound on the surface.

DR. CHOU: Thank you.

DR. ROBERTS: Dr. Mushak, then Dr. Solo-Gabriele, then Dr. Styblo.

DR. MUSHAK: Two quick questions. One is: Did you do a before and after surface analysis with the surface methodologies? I mean, it's important to know what came off in a quick and dirty scrub. But it's also important to know what stayed. And this is a takeoff on Dr. Chou's question.

Second one is if I might just quickly posit them. Artifactual interconversation of chromium forms based on the laboratory methodology, was that a problem? You mentioned very low chromium 6 levels. Could that arise from simply the extraction conditions?

DR. KAMDEM: Thank you for your question. Yes, for the surface characterization, what we did, first, we used untreated wood for our background. Then after washing and brushing, we went again and redo the chemical analysis. And we find almost the same, the different picks with XRD and the XPS. But you have just a difference in term of intensity. And so just in that you have some fixation on the surface. If you wash, you still have some chemical on the surface.

DR. MUSHAK: And the interconversion of chromium by methodology?

DR. KAMDEM: We did a lot of study. And there's a lot of study in the literature regarding the characterization of chromium 6 to study the fixation of CCA-treated wood. And there's a lot of

parameters in the extraction. And I believe that this method, the diphenicarbozide method, is very sensitive. With this method, you can detect something like 1 ppm of chromium 6.

So I don't think that if you did not detect the chromium 6, it was a problem of extraction. I think that the chromium 6 was just not there on the surface or in the water that we used to wash the wood deck.

DR. ROBERTS: Thank you. Dr. Solo-Gabriele.

DR. SOLO-GABRIELE: I have a related questions that were asked earlier.

You mentioned the name of the treating company that treated the wood. What was the method of fixation? And, also, what was the time elapsed between the time the wood was treated versus the time that the planks were analyzed? And I have a couple of extra questions.

DR. KAMDEM: I did all the analysis, the Great Southern Treater didn't send me any analysis. I did myself the analysis. And, again, I assumed that the sample that the deck that was sent to me was at least 16-months old from the treatment.

DR. SOLO-GABRIELE: Do you know the method of fixation? Did they just let it sit there?

DR. KAMDEM: Well, there are several methods of fixation, but I don't know which one they used.

DR. SOLO-GABRIELE: They're dried.

DR. KAMDEM: Yeah, it can be air dried or it can be also steam condition to increase the fixation. I don't know what they used.

But still I assumed that usually in the wood CCA treatment after three months, we think that after even 48 hours for a laboratory sample, you have like 99-percent fixation, usually with a small cube, the three-quarter inch cube. And also for the treatment, usually we did it 48 hours. You would expect the best management practice that is advised that is put forward by the AWPA. Within 48 hours in the south, you have a fixation complete.

DR. SOLO-GABRIELE: The other question I had was related to the pH of the DI water generally doesn't have very much buffering capacity. Was this the pH before you washed the plank or after?

DR. KAMDEM: No, before, before.

DR. SOLO-GABRIELE: Before. Did you measure it after?

DR. KAMDEM: No.

DR. SOLO-GABRIELE: Did it change?

DR. KAMDEM: No, no. No, we didn't measure the pH after we washed.

DR. SOLO-GABRIELE: And the atomic absorption method for arsenic analysis, what was the detect limit?

DR. KAMDEM: The detection limit is around 10 ppm.

DR. SOLO-GABRIELE: 10 ppm.

DR. KAMDEM: Yes, for AAS. You can go lower than that, but I always want to be very conservative. I would say 10.

DR. ROBERTS: Thank you. Dr. Styblo, then Dr. Steinberg.

DR. STYBLO: Yeah, two questions. First, was any part of the wood you analyzed exposed to soil? I assume it was exposed to other environmental media.

Second question: The method or methods you used for arsenic speciation, were they able to analyze, detect methylated organic species of arsenic? Did you, for example, use any organic arsenic as standard? I haven't seen it on the chart.

DR. KAMDEM: Thank you. Usually for the deck, they are what we called the "above ground." That means the deck is not in contact with the ground. You can have only the post that after that different retention. I don't think that the wood deck that we studied was in the ground contact. That's the answer for the first question.

Now for the second question for the speciation of arsenic, no, we didn't use any organic arsenic. We just used -- for the calibration

and for our test, we just used arsenic III and arsenic V, the oxide formulation.

DR. STYBLO: Can you exclude that organize arsenic was represented?

DR. KAMDEM: No. Because again the study just showed that to have arsenic V. We may have arsenic V methylated.

DR. STYBLO: Well, could be it methylated arsenic V?

DR. KAMDEM: That's what I said, maybe. But we have to go back and do more study. But, again, the XPS is based on the chemical shift. And usually the method is very difficult to shift the electron from the chromium. It would be a little bit more difficult to do that with methylated arsenic. Maybe the best way to do it would to develop an ICP or HPC metal for that.

DR. STYBLO: Thanks.

DR. ROBERTS: Dr. Steinberg and then Dr. Francois.

DR. STEINBERG: In your paper you say that X-ray defraction is a useful technique for investigating the compounds with ordered structure. Then you say it can be used to identify and semiquantify chrystaline compounds present in a matrix. Also, you list six different techniques that can be used for measuring different metals.

Have all of these standards been correlated, for example, with

gold standards like atomic absorption? Could you help clarify that a little bit?

DR. KAMDEM: Yes, I will try. I would say, yes, you can use X-ray defraction to get an idea to know if you have a crystal or an amorphous solid. Now, if dependent on the size of your solid, because X-ray defraction detection is not like the same thing you have with the AA, which is based on element, X-ray defraction is mostly based on the particle size of your -- not particle size -- the crystal that you have, which is defracting the light.

So I would say, yes, you can do that. But we haven't done that yet. That's why we say semiquantify. We haven't done any correlation.

We have some correlation with CDDC, which is a different wood preservative. But we're lucky because with CDDC, copper dimethyldithinocarbamate, you have a very nice defraction pattern. But it's not true for all the other crystals that you would find in real life. So that's why we say semiquantitative metal.

DR. ROBERTS: Dr. Francois.

DR. FRANCOIS: I just wanted to know if the Panel had received copies of these slides?

DR. ROBERTS: I don't. We have? Okay. Thank you. Dr.

Ginsberg and then maybe we could move on to the next speaker.

DR. GINSBERG: Yeah, I found this to be a very interesting study because I hadn't seen anyone else try to characterize what this dislodgeable material actually is, both with physical means and also with some chemical means. And I guess I'm interpreting your results in terms of the percentage of arsenic that's present in the solid weight of material that's there, this rotovaped material. You said it was .2 percent, which converts to 2,000 parts per million.

Now, I don't know if you got a lot of splinters, you know, actual solid wood pieces in there that would tend to create artifacts. But if this is mostly dirt, so to speak, that's on the surface of this wood as you washed it and did some scrubbing, if that's what we're talking about that's rotovaped down and at .2 percent, then we're talking about a dislodgeable residue that's about 2,000 parts per million with respect to arsenic.

So I just want your input in terms of what does the solid residue represent in your study?

DR. KAMDEM: Thank you. Actually, I have a slide showing the microscope of some residues. There at end, yes. That was the ESEM. Next, please. Yeah. Right here.

See. This is a piece of wood. Right. And this may be why you

have such a high level of arsenic still in the solid residue. You can see here the lumen. Those are fibers here, structure there.

And just to show just the size of the glass wool that I used for filtration. And you can see here that those are piece of wood residues.

DR. GINSBERG: So then what the chemistry data that you were presenting isn't necessarily for what we would think about as dislodgeable arsenic, but it's a combination of solid pieces of material wood splinters that might be picked plus dislodgeable. Is that the way to interpret your data?

DR. KAMDEM: No. The solid that we removed with the glass-wool filtration before the rotoevaporation.

DR. GINSBERG: I'm sorry. Repeat that.

DR. KAMDEM: This is the solid that was removed with the glass wool filtration before the rotoevaporation, yes.

DR. GINSBERG: Oh, okay. So then my first thought was the right direction.

DR. KAMDEM: Yes, yes.

DR. ROBERTS: Dr. Matsumura has one final question.

DR. MATSUMURA: Just a quick one.

Did you look at the rotoevaporation product? What did you remove? I mean, you have to heat it up to get the concentrating;

right? So it's water. So the question is: Is there any volatile components which is codistilled with the water?

DR. KAMDEM: Thank you for your question. For rotoevaporation you use vacuum and then you collect all your evaporated product. So there's nothing lost. And then after, we did analyze the water that was collected.

DR. MATSUMURA: There should be two components that you concentrated, then evaporated, then reconcentrated.

DR. KAMDEM: Yes.

DR. MATSUMURA: Which one did you measure?

DR. KAMDEM: Both in the solution that was rotoevaporated for concentrate was analyzed and also the water. That water was removed through the vacuum and temperature was also analyzed. And there was nothing. No copper chromium arsenic detected.

DR. MATSUMURA: All right.

DR. ROBERTS: Thank you, Dr. Kamdem, for your presentation. One really, really short question.

DR. SMITH: I want to just make sure I understood this figure.

So what you're saying is that the brushing with the water removed material that collected on a glass-wool filter revealed presence of particles that are wood fibers in nature; is that correct?

DR. KAMDEM: Yes.

DR. ROBERTS: Thank you, Dr. Kamdem, for your presentation and answering our many questions.

Dr. Aposhian, I'm going to let you take us up to the break.

DR. APOSHIAN: My name is Vashen Aposhian. I'm a Professor of Molecular and Cellular Biology, Faculty of Science, and Professor of Pharmacology and Medical School at the University of Arizona.

I've been asked to present to you the bioavailability studies. So anyway, I've been asked to present the bioavailability studies that we have done on dislodgeable CCA.

What I'd like to do during this brief presentation is to first review with you the metabolism of inorganic arsenic in the human being. Second, I would like to address the question as to what is the best animal model to study.

You said you'd put the lights out so that it would be more visible. Thank you, Johnny. Is that in focus for you all?

So, second, I would like to discuss with you the question as to what is the best animal model to study the metabolism or the bioavailability of an inorganic arsenic.

And then, finally, I would like to present our results, both the

bioavailability studies and the distribution, in the liver and the kidney of inorganic arsenic.

Let me make it very clear that we have just been following the arsenic. We have not been following the chromate. We have not been following the copper. We have used the arsenic as a label. And we have just been studying this problem for less than 45 days. The research I'm going to tell you about has been supported in part by the University of Arizona, the Osmose Company, and the Arch Chemical Company.

Now, unfortunately, this is not as visible as it could be. But let me go over it with you very quickly.

Arsenate to arsenite, we have recently purified and sequenced this enzyme. The liver cell has tremendous capacity for this conversion of arsenate to arsenite. And Dr. Mushak was very, very correct when he said that you can't separate the toxicity. There is tremendous capacity in the cell for this conversion.

The arsenite is then methylated. It was going to be methylated. And then it is reduced to MMA3 and then further methylated again, reduced to DMA3 and so on.

Now, our laboratory and Dr. Styblo's laboratory have probably spent the last six, seven years studying this pathway. And I could say

fairly, the results of both our laboratories clearly show that this is not a detoxification procedure. Classically, methylation of arsenic has been called a detoxification procedure, but it is not so.

In our laboratory and in Dr. Styblo's laboratory, we've shown that this, what we call methyl MMA3, that MMA3 is more toxic than inorganic arsenic -- inorganic arsenite. And Dr. Styblo's lab along with Mark Mass have shown that dimethyl, or DMA3 as we call it, is able to cleave DNA. It's the first time that there has been a chemical reaction that I know of that has been shown to occur between an arsenic compound and DNA.

So I don't want -- please don't leave the room thinking that this is a detoxification procedure. We consider this to become a more toxic compound.

The advantage of methylation is that it does increase the excretability of an arsenic compound. But during that process to increase excretability, you're making two more toxic compounds.

The other reason I'm showing you this slide is for any animal model to be pertinent to the human, if a speciation study has not been done as far as what's coming out in the urine, then that study has to be questioned. Not all animals will methylate inorganic arsenic.

This slide that one of the my graduate students made up, which I

think he got mostly from Marie Vahter's work, quite frankly. This is the human. This is inorganic arsenic. The white is MMA, and the red is DMA.

Now, let's go over the rat. The rat for three reasons is not considered to be a good model to study arsenic metabolism. One reason is that its biliary excretion of inorganic arsenic is the highest of any species known. The second reason is that the DMA binds the red blood cells of the rat, and this DMA binds with such a tenacity that it's not seen in any of these other animals to that great extent. And, finally, you can see that the white area here, the MMA, is much less in the rat than it is.

Now, let's take the chimpanzee. The DNA of chimpanzee, not the DMA, the DNA, the deoxyribonucleic of a chimpanzee, is 99-percent similar to that of a human. There is no other species as close to human as far as its genetic material is concerned as the chip is. But Marie Vahter clearly showed that the chimpanzee, when challenged inorganically, will not excrete any methylated arsenicals.

In our laboratory, we're very fortunate to get livers from chimps, and we cannot detect any methyltransferases. So I think the current opinion is chimp cannot methylate. Okay. This is also true with marmosets monkey; to some extent, the guinea pig.

Let's now go over to what I and Marie Vahter and many others consider to be the best model for studying arsenic metabolism as compared to human. And that's the rabbit and the hamster. And you can see the white areas are not as much, but certainly closer. And they're very easy to work with.

The dog is not on here. And if you go through the literature about bioavailability of inorganic arsenic, you'll see these old studies about the dog. The dog does not put out any MMA at all in the urine. So, again, it questions what species should you use.

This shows you the efficiency of arsenite methyltransferase activity among nonhuman primates. I've told you about the New World monkeys. We've gotten livers from all of these animals. And only the macaque seemed to put out methylated arsenicals in the urine once they're challenged.

The great ape, the gorilla, we wanted a liver. I wanted to talk to my friends at the San Diego zoo. I wanted to go in and get gorilla urine after they shot a dart with a tranquilizer. And the head of research there said, no, I was too old and might not be able to run fast enough. He suggested I get one of my graduate students. But since my graduate students are among the best in the country, I just did not want to endanger them. So we don't have urines here. But as you can

see, most of the monkeys don't seem to have the enzyme.

For those of you who don't know how such studies are done, this is a metabolism cage. It's a cage made of plastic with just a metal screen, and these are food and water bottles. And there's a device here to separate urine and feces as they fall. And here you can see this is the urine bottle and here is the feces drop. And we put a fiberglass screen on this so that we don't get any feces into this preparation.

How do we do these experiments? So we picked the hamster first of all. And we consider the hamster, for many reasons, to be a good model for bioavailability protocol.

We took dislodgeable CCA that was sent to us and diluted it to 16.5 micrograms of arsenic, gave it in on -- it was actually 16.5 micrograms in 0.15 mils. of water -- gave it by gavage to three male hamsters and three female hamsters. We tried to abide by the NIH rules asking for equal representation of the sexes.

Because the problem is the female is just not as consistent in the data because sometimes one or two of them are in estrus and we have that problem. But the males give us very consistent data, but we still use them both.

Control gets doubled-distilled water by gavage. And the

controls were two male hamsters and one female hamster.

These are considered to be young hamsters. We start them about 75 grams when we begin with them. I don't know how that relates to -- I used to know how it related to a human age, but I have doubts about those numbers.

We collect the urine and feces for one control day and each of five days after gavage. So we could do 24-hour urine and 24-hour feces determination, total arsenic analysis of urine, digested feces, and digested tissues by ICP mass speck.

Those of you who don't have an ICP mass spec, I urge you to get one. It has made AAS, atomic absorption, out of date. We can detect 0.05 nanograms per mil or those of you familiar with liters, 0.0359 micrograms of arsenic per liter, which is very different. And we were finally able to convince our vice president for research to get us one, and it's just absolutely wonderful. You do a urine in less than one minute. You absolutely don't have to digest it. You have to digest the feces. Anyway, analysis of urine species by HPLCICP mass spec can also be done.

Mass balance first. We gave 16.5 micrograms of arsenic as dislodgeable CCA by month. We recovered 15.6 micrograms arsenic was recovered as mean urinary arsenic and mean fecal arsenic. So we

were able to recover into the five-day period 95 percent of the arsenic that was given. And we're very pleased with that number.

These experiments take a tremendous amount of time to wash down one of these metabolic cages. In a quantitative way where you want to keep your volume of water very small, it takes about 45 minutes. I don't have the patience to do it myself, but my people have a great deal of patience to do it.

So the bioavailability, the formula that I use that some people use, but I'll talk about that in a minute. Urinary arsenic totaling -- fecal arsenic times 100 other urinary arsenic over ingested arsenic. We have done both. Figures are within 1 or 2 percent of each other. It really didn't...

This shows you the -- I'm showing you crude data here, the bioavailability of dislodgeable CCA in hamsters. We name our hamsters because you make less of a mistake if you write down Anna or Betty than if you write down one or three or four. And so all our hamsters have names.

These, the bioavailabilities, as you see with Carl, Doug, and Ed. The males are quite close. The women -- I'm sorry -- the females have a little more variability. We rejected Ed, one of our best bioavailabilities, because he was excreting much more arsenic than we

gave him.

Don't laugh at this. You've got to understand animals. This particular hamster ate his feces. All right. So we give him liquid diets. It is not unusual for rats, mice, or hamsters to eat feces. You've just got to be able to observe them well enough to know it. And so, therefore, we've rejected that data. So our mean, excluding Ed for bioavailability, was 11.4 percent.

This shows you the plot. I apologize again for the smallness of the letters. On the left-hand side is, I think, nanograms of arsenic per 24-hour period; and on the bottom is the date that it was done.

We looked at the livers and kidneys of hamsters in the same experiment. After the five-day period, they were euthanized with CO2, the livers and kidneys taken out, blotted, cleaned, washed. What we usually do. And then analyze digested and then analyze by ICP mass speck.

The point I want to make is there is absolutely no significant difference between these numbers, 15.9, 12.4. The P was greater than 0.5. The P was actually 0.45. So maybe with more animals, this may become significant. But I sort of doubt it. They are not significantly different.

Now if I can have that -- Johnny, if I can have that. Thanks.

Oh, it's not Johnny doing it. Okay.

Again, I want to apologize for this one. I stopped on the way to the airport, at the lab, thinking it would be handy for you. You've got to do -- I humbly suggest that if you're going to do bioavailability studies, you've got to do the whole thing in the same lab. You've got to know what the compound, what the absolute bioavailability of the compound is you're studying. And if you're going to compare it to something, you've got to have that comparison done in your laboratory.

And the reason I say this is this is a paper by, I think, Charbonneau, 1980. And on top is Marie Vahter. And most of the people that are arsenic people, Styblo, Kosnett, Hopenhayn-Rich and others here, know Marie Vahter; and she's a very reliable investigator. But note that with arsenate, As(V) now, that she got -- both of these are with hamsters. The amount in the urine, if I can see correctly, was 74.7 percent. That's the amount of arsenate, soluble arsenate, in water given by mouth that 74.7 percent came out in the urine.

Now, if you go down to the Chabineau's study, 70 percent. Same kind of experiment, same animals. Seventy percent was found in the feces. One of these reasons, I think, is Chabineau used 0.01 micrograms of arsenate. You know, that's so small that it could get

lodged on a hair in the trachea or something and you'd never see it. Or the other way is it wouldn't be absorbed.

So, again, I would like urge the Committee to consider studies that have been done where the absolute values they've come up with those values are from the same lab. And I, also, hope that the Committee has gotten a paper written by Dr. Roberts which, I think, was a technical paper submitted to the Florida Department of something. Again, I meant to pick that up, and I forgot to bring it with me.

And in there he says, quite frankly, that the use of animals to bioavailability studies as to what the best model is is very difficult to answer. I'm not using his exact words. I had hoped to have it on something like this.

Now, if I could have the last slide which I hope is the summary. They're we are.

Summary. There should be concern about the appropriate animal modeling for inorganic arsenic. The bioavailability for dislodgeable CCA is 11.4 percent plus or minus 1.8 percent, the mean, using hamsters. There was no significant difference in liver or kidney arsenic concentrations for dislodgeable at CCA-treated animals versus control animals.

Thank you for your attention.

DR. ROBERTS: Thank you. Are there any questions for Dr. Aposhian? We have Dr. Mushak, Dr. Kosnett, Dr. Bruckner. Dr. Mushak first.

DR. MUSHAK: Two quick questions, Vas.

How young are your young hamsters? You know, the method section said "young hamsters."

DR. APOSHIAN: We receive them when they're 75 grams. They are about five to six weeks old. That's about all I can tell you. That's all I know.

DR. MUSHAK: I'm just trying to get a developmental idea of where they are on the comparability spectrum.

The second one is on the fecal portion of the arsenic. How do you break out endogenous fecal versus unabsorbed?

DR. APOSHIAN: We have the controls.

DR. MUSHAK: Yeah.

DR. APOSHIAN: And what we've done is we've subtracted the daily mean fecal control arsenic from the experimental ones. And we think that's a reasonable kind of correction to make.

DR. MUSHAK: Yeah. But I don't know that that gets you out of the box.

DR. APOSHIAN: In what way? I don't understand.

DR. MUSHAK: I mean, proportion to the dose, you know, a certain fraction is going to be endogenous and that's going to increase as you ratchet up whatever the dose is. I don't see how control permits you to break out that. You almost have to do this by double isotopes or something.

DR. APOSHIAN: Again, all I can say is that if you set up certain parameters, the ones that we set up, and are certain that the control animals are treated exactly the same as the experimental animals with the exception the controls don't get any added arsenic in the diet, that takes care of the problem. I hope.

DR. ROBERTS: Dr. Kosnett.

DR. KOSNETT: Thanks for presenting that, Vas.

What do we know about the biliary excretion of arsenic in the hamster? And if that was a significant portion, how might that affect the interpretation.

DR. APOSHIAN: Curt Klossen published a classical paper, I think, 1985, in which he goes through a number of species. I don't remember the exact number, but I remember the rat being the highest. And I remember there was not anything unusually high about the hamster. I don't have the figure right at my fingertips. I'll try to send

you that. I have to leave tonight. I'll call you tomorrow. Are you staying in this hotel, Michael? Okay.

DR. KOSNETT: Or e-mail the data.

DR. ROBERTS: Dr. Bruckner.

DR. BRUCKNER: I was just curious. You mentioned the word "toxicity" several times, and you made the point that methyl was more toxic than inorganic. I'm not sure what you said about dimethyls as opposed to monomethyl. What do you mean by toxicity?

DR. APOSHIAN: Okay. Toxicity data are a combination of experiments that were done in my laboratory and Dr. Styblo's laboratory. In our laboratory, the tissue culture experiments were done based on potassium leakage, LDH leakage, and I've forgotten the term -- there's a dye that we use for mitochondrial damage. All right. These are classical toxicology parameters that are used. And I don't remember what Miroslav used for his tissue culture ones. I think there was a cytotoxicity.

DR. STYBLO: Well, we used the mitochondrial dye, MTT.

DR. APOSHIAN: In addition to that, our animal committee, which doesn't like LD50 studies, allowed us, because they thought the problem was very important when we were doing this with some other compounds, to do LD50 using hamsters. So it's a straight lethal dose

50 kind of experiment. That's what the data on MMA3.

On DMA3, Dr. Styblo and Dr. Mass, Mass working for the EPA down at Research Triangle, has done a number of experiments that, first of all, neither -- and correct me, Miroslav, if I'm wrong -- that neither arsenite or arsenate damaged DNA, a Fix DNA, a double-stranded DNA, in an in vitro assay.

But when they added MMA3 or DMA3, there was cleavage, if I remember correctly. And they also did, I think, a lymphocyte experiment which also showed, if I remember correctly, that DMA3 was the most toxic of all the arsenic species.

Does that answer your question?

DR. ROBERTS: I have one. Then Dr. Hopenhayn-Rich has one and Dr. Ginsberg.

Dr. Aposhian, could you comment just a little bit on the relationship between metabolism and absorption. And when we do species comparisons in terms of metabolism, it's obviously important when we're trying to do toxicity studies. Why would necessarily species that metabolizes arsenic similar to humans have absorption similar to humans? Can you explain the connection for me?

DR. APOSHIAN: I think this will answer your question. Arsenite has such a PK value that at body pH it has no charge. So

arsenite usually diffuses right through. There's no carrier mechanism for getting arsenite into a mammalian cell that we know of.

Arsenate is taken up by the phosphate carrier mechanism. Arsenate gets into cells in the kidney, cells, and other things by the phosphate carrier mechanism. What brings them out, again, this is -- yeah, it just appeared. Suzuki in Japan has shown that what gets out of the cell is -- well, what the blood brings to a cell, to a liver cell, is DMA3. In the cell it's converted to DMA5, and what effluxes from the cell is DMA5.

Now as far as why metabolism is important for the comparison is that if you're not going to methylate -- we methylate. All right. But if we compare our arsenic processes to an animal who doesn't methylate, it's sort of like comparing apples to oranges almost.

There is a big difference. Does that answer your question, Dr. Roberts?

DR. ROBERTS: Well, sort of.

DR. APOSHIAN: Perhaps you could be a little more specific.

DR. ROBERTS: Well, empirically -- we can have this discussion perhaps off-line another time. I think that there's other issues about selecting models in terms of empirical comparisons of how that animal handles and excretes arsenic that are useful for

selecting animal models. And I won't take up the Panel's time. Maybe we can discuss that at another time.

Let me see. I had Dr. Hopenhayn-Rich and then Dr. Ginsberg.

DR. HOPENHAYN-RICH: I have two questions, and they also relate to toxicity and the choice of the model. One is if the hamster is considered the best model in terms of similarity of the distribution of metabolites, is there a difference or could there be a difference between the amount and the duration of MMA3 and DMA3 in the hamsters versus the humans? You compared the proportion of just MMA, DMA, and inorganic. I'll just make that the two questions.

DR. APOSHIAN: I think we're the only people. I could be wrong. No, I think Suzuki also may now have a paper out.

We are the first and probably the only people who gave radioactive arsenate to hamsters and took their tissues out and looked at what was in the liver. And we found that there was almost an equal amount of MMA3 and MMA5 there.

Okay. We have been trying to get human livers to do this. The problem is we have to get human livers from a place where they've been exposed. Goamazumba has offered us some. And it's just a matter now -- in fact, Michael Kosnett brought us back some livers from Masumba maybe three years ago. Michael, are you going again?

Because we need someone to bring it back. My wife won't let me leave the country at the present time.

DR. ROBERTS: You might not want to discuss this in a public forum.

DR. APOSHIAN: So that's about all we know.

DR. HOPENHAYN-RICH: The other question is that I wonder how, you know, we've heard repeatedly here and in many other places where arsenic has been discussed, that there is no good animal model for the toxic or long-term effects of arsenic, the cancer effects, et cetera. And I wondered what is the relationship, then? If the hamster is a good model for methylation, how does that relate to the lack of a good model for cancer?

DR. APOSHIAN: I'd like to correct you. No one says that there's no good model. There are good models.

DR. HOPENHAYN-RICH: Yeah, I didn't say that today; but other people did.

DR. APOSHIAN: There are good models. And I think more and more, the cancer models are becoming more acceptable, some in Australia as John Abernathy mentioned. Toby Rossman has one. Perhaps Dr. Styblo -- I didn't get to the Sardinia meeting. Maybe Dr. Styblo could tell us if he remembers it.

But there are now and certainly Michael Wackus at the NCI-NIHS has, again, another fairly decent animal model. But all of these have not stood the test of time. There's been a tremendous explosion of interest and money to study this. So I think we are going to have good animal models for carcinogenicity.

DR. HOPENHAYN-RICH: But is the hamster, the same species that you used for this methylation, for this study that you're saying compares well to the human in terms of methylation. Is this hamster a good model for our cancer studies or other human endpoints?

DR. APOSHIAN: I don't know. We're just studying bioavailability. We have a research grant that we will study other things in time. But right now, we have just studies the bioavailability. So I really can't tell you whether it's a good model for carcinogenesis at this present time.

DR. ROBERTS: Dr. Ginsberg, do you have a final question for Dr. Aposhian?

DR. GINSBERG: I guess a fairly simplistic interpretation of your data showing low bioavailability would be that the form of the CCA-derived dislodgeable material had the arsenic in some kind of complex or insoluble state that didn't get absorbed well into these exposure conditions.

Do you know what the pH is of the hamster gut in terms of dissolution perhaps of such a CCA-complex? And do you know if -- I didn't catch it. I think you said it. What the dosing volume, the gavage volume, was then it went down and whether that volume might affect the pH entering the gut.

DR. APOSHIAN: The volume was 0.159 milliliters.

DR. GINSBERG: What is that per kilogram body weight?

DR. APOSHIAN: Excuse me?

DR. GINSBERG: What's the volume per kilogram body weight, if you can

DR. APOSHIAN: Divide 0.15 mil by approximately 100.

DR. GINSBERG: Okay.

DR. APOSHIAN: And that's what you get. You had a -- what's the first part of your question?

DR. GINSBERG: What's the pH of the gut?

DR. APOSHIAN: I don't know. That's a good question. When I get back, we have some extra hamsters to check that out. My guess is that it is probably pH 1 to 2 of the stomach.

DR. GINSBERG: Well, rats are like 4, 4-ish. It depends upon fasting and fed. Were these fasted animals?

DR. APOSHIAN: Before they were given the arsenic, they were

fasted overnight, yes.

DR. ROBERTS: Thank you, Dr. Aposhian, for your presentation.

We want to do one more short presentation before we go to break. Mr. Feldman has a short presentation. Then we're going to take a 15-minute break.

MR. FELDMAN: Thank you. I appreciate the opportunity to comment on your work on this important issue.

I'm Jay Feldman, Executive Director of Beyond Pesticides National Coalition Against the Misuse of Pesticides.

I wanted to first start before getting into some of the specific comments on EPA issues and questions and just put some context to this discussion. We've been working on this issue since the early 1980's. But as you know, EPA has been working on this issue since it initiated an ARPAR in 1978 and in a special review.

And I believe that the context of this has resulted in continued exposure to heavy duty wood preservatives that has caused a silent tragedy because of EPA's failure to act on the side of caution, its failure to embrace a precautionary principle for protection of children, and failure to enforce the unreasonable adverse effect standard under FIFRA.

We would not really be here today if EPA fully embraces its statutory standard, acknowledged the continuing failure of voluntary risk mitigation measures, recognizes the full extent of contamination and poisoning caused by inorganic arsenicals from wood treatment to use disposal, considered worker hazards, and treatment site contamination, and evaluated the substitutability of wood preservatives with nonchemical alternatives.

EPA acknowledges in the purpose statement for today's meeting that the issue of children and exposure to playground equipment has been hopped up in the queue as a result of public concern, which right on the face of it, really does acknowledge the politicized nature of this process. In fact, parents and media outlets have found 25-times background levels in studies looking at arsenic and soil around equipment, playground equipment.

But what's most troubling here from our standpoint is that there is no apparent urgency to this process on the part of EPA. We believe that EPA and SAP needs to recognize the eminent hazard associated with continued exposure to CCA and other heavy duty wood preservatives. The situation is made even worse by the fact that for virtually every wood preservative use there is an economically competitive less or nontoxic alternative.

Now turning to the EPA issues, I just quickly run through some of these. In looking at the endpoint selection for arsenic, certainly EPA should apply a tenfold margin of safety for childrens's exposure given the sensitivity to this population group and in setting the acceptable margin of exposure should clearly recognize a full range of dietary, nondietary exposure, backgrounds levels, and their geographic variability, water levels, indoor and outdoor ambient air levels. With these exposures taken into account, there is very little, if any room, for additional exposure.

In terms of bioavailability, in addition to the discussion you've been having on appropriate species for testing, we believe you ought to look at the bioavailability based on different soil types, including the full range of soils with high and low organic matter.

In terms of dermal absorption, you should take into account the condition of the skin, abrasions, cuts, all of which affect the value of dermal absorption. And in addition to that, you should consider an injection exposure to anyone that has played on playground equipment or backyard deck knows that the possibility of a splinter exist. Splinters can mean that chemical residues enter the blood stream, and EPA cannot ignore this exposure scenario.

Hazard characterization, EPA should look at the worse case

scenario, new wood that is not fully fixed as it does recognize in the purpose statement today endpoint selection of chromium 6.

Regarding chromium 6, why is it that EPA recognizes chromium 6 as a known human carcinogen for inhalation but is prepared to discount all exposure by the oral route because it, quote, end quote, "cannot be determined whether it's a carcinogen."

The Agency must consider all possible routes of exposure and the resulting effects. Certainly, ATSDR and its toxicological profiles, which I don't, on the surface anyway, see referenced by EPA, certainly create the data base necessary to look at this route of exposure for chromium and arsenicals.

In endpoints for dermal risk, again, to dismiss systemic effects from dermal exposure as irrelevant, we believe flies in the face of, again, the ATSDR toxicological profiles with findings of systemic effects associated with dermal exposure.

In terms of the methodology for characterizing childhood exposure, in calculating exposure, the full range of background levels, as I said earlier, dietary, nondietary, must be considered including air, water, food, decks, park benches, picnic tables, medical applications and other exposes.

And in its risk assessment, EPA must disclose all the

uncertainties associated with its assumptions. Since the distribution the Agency chooses has associated assumptions, those assumptions must be disclosed and the Agency must perform a sensitivity analysis of its model explaining how sensitive the model is to various assumptions and explaining how different the outcome would be if different assumptions were used.

Under the Agency's risk cup approach, it must be clearly stated what contribution these exposures make to the overall acceptable exposures defined by EPA. EPA must aggregate this with other nondietary and dietary risks that children and the general public assumed to have.

In terms of transfer of residues, the Agency must assume that residues taken from wood surfaces to skin or from soil to skin spread to numerous sites of the body. It cannot be assumed that only one hit of a dermal chemical exposure is associated with one touch to the wood or soil. In fact, there are numerous touches and, therefore, numerous dermal exposures associated with the touch of a treated-wood surface or contaminated soil.

Skipping down to inhalation variability, the Agency cannot assume as it has stated, that inhalation potential from contact with either CCA-treated wood or CCA-contaminated soil is negligible.

Certainly, inorganic arsenicals attached to soil particles kicked up as dust can be inhaled or the ingested. The Agency has a lot of history looking at pesticides in dust, and it cannot assume that CCA does not behave in a manner that results in contaminated dust.

In terms of buffering materials, EPA must immediately outlaw the practice of creating wood mulch products from CCA-treated or other heavy duty preservative-treated wood. The concentration levels are unacceptable, and the threat of children picking up tainted wood and putting it directly in their mouths is great. This is a no-brainer and should be adopted by the end of this afternoon.

In terms of sealants, this is a short-term transition tool. Sealants are not a long-term solution. EPA cannot control the process by which sealants are applied, the certainty that it will perform a risk mitigation measure.

And then, finally, when EPA evaluates CCA, it cannot confine its review and analysis to only arsenic chromium and copper, rather the agency must look at all the biologically and chemically active constitutes contaminants and ingredients in the CCA formulation. Otherwise, you have a false outcome from your risk assessments with false assumptions.

Thank you very much. Again, I appreciate the work that you all

are doing individually and collectively and the guidance that you give to the EPA.

DR. ROBERTS: Thank you, Mr. Feldman. Are there any questions for Mr. Feldman on his comments or presentation? Yes, Dr. Wargo.

DR. WARGO: I'll be brief. I know everyone is anxious for a break.

A couple of points. You suggested, Jay, that you think that the legal standards that are embedded in the Food Quality Protection Act, specifically the tenfold safety factor and the need to do aggregate and cumulative exposure assessment, apply in this case.

MR. FELDMAN: Yes.

DR. WARGO: I apologize, by the way, for coming in late, and perhaps this was covered earlier.

I'm wondering whether or not this cumulative exposure issue is on the table.

DR. ROBERTS: It's been mentioned a couple of times.

DR. WARGO: Is that part of the Agency's perception charge here to understand cumulative exposure?

DR. ROBERTS: Yeah, I believe the Agency explained a little earlier today that this is really one step maybe out of the normal

sequence of steps that would be involved in risk assessment at a variety of levels that would include cumulative exposure.

DR. WARGO: Okay.

DR. ROBERTS: Did I do okay on that?

DR. WARGO: Also, you may have covered this question earlier. And I know, Jay, you in the past have done some work on this.

The documents that I was sent by the Agency didn't give me a sense of the magnitude of this issue in terms of total amount of CCA that's produced per year in the U.S. And I, also, don't know what percentages of, say, even the soft-wood supplies in the U.S. are pressure treated. And, also, I'm not at all clear about what happens to this stuff once it ends its useful life, whether or not it is dumped in landfills. And you made a reference to it being chipped up as mulch.

And so any kind of basic statistics we've got to understand the scale of this issue would be quite helpful to me.

MR. FELDMAN: Yes, actually, the EPA relies on the statistics collected by the American Wood Preservers Institute for overall poundage numbers.

You know, the use of wood preservatives -- this includes all the three heavy duty wood preservatives -- collectively equal about half

of all conventional pesticides used, taking out chlorine and disinfectants. That's an extraordinary number which is supported by the AWPI data base on volume of use.

You know, I'm sure EPA can give you more exact numbers on that, but we're talking in the area of a billion pounds a year or close to it. And, certainly, I think, John, you have a lot of experience with the issue of cumulative risk or additive risk.

And certainly it's our position that for the SAP or any deliberative scientific body to fully evaluate the risk to children, one would have to fully evaluate the life cycle of the wood preservative from production through disposal, given that we know, certainly, that EPA has not regulated a wood taken out of service. And so it does end up in community landfills and does then create a potential contamination problem that affects the overall toxic body burden as a result of potential water contamination, ambient air contamination, and other sorts of contamination associated with disposal in unlined municipal landfills.

So I'm glad to hear. I also was not able to be here earlier, dealing with the anthrax problem in our local postal service. But the issue of looking at this in the context of FQPA and the statutory mandate to evaluate multiple exposures, aggregate risks, interactions

perhaps between chemical, certainly chemicals that have the same common mechanism of effect which we're dealing with here is critical.

DR. ROBERTS: Any other questions before we go to break? If not, let's take a 15-minute break. We have a number of very important presentations yet to do. So please hurry back, and let's try to start promptly in 15 minutes.

[Break.]

DR. ROBERTS: Before we proceeded with the next public presenter, Dr. Vu would like to offer clarifications on an issue that came up just a little while ago. Dr. Vu.

DR. VU: Thank you, Dr. Roberts.

We would like to clarify some of the definitions that were brought up earlier by the public commentor that we had before the break; and, also, Dr. Wargo raised the issue cumulative risk and aggregate risk, et cetera.

The Agency has defined cumulative risk to mean that the risk associated with combined exposure from multiple stressors. And multiple stressors could be defined as chemical agents, biological agents, and physical agents. So really, cumulative risk is risk with multiple stressors.

Under the FQPA law, the cumulative risk has been defined

much more narrow of focus. It refers to risk associated with pesticides that have common mode of actions.

For example, the Agency and Office of Pesticides Program is conducting preliminary risk assessments of organophosphates which have common mode of action associated across all the 24 organic phosphates and that risk associated with it. That's the one activity. That's the cumulative risk defined under FQPA.

Aggregate risk is defined as risk associated with a single stressor, whether it's a chemical or biological agent, et cetera, cut across all sources of exposures and pathways.

So if you think about CCA products, the risk associated with all of these sources of exposure, whether it's from a life cycle that would be more of an aggregate risk.

So with regard to whether the Agency is going to consider aggregate risk with regard to CCA, the Agency will certainly consider aggregate risk when it makes sense or is applicable. So that's the issue at the table.

And the second issue raised was with regard to childrens's risk. Certainly, the Agency would look into the exposure dose, as well as the susceptibility issue on inherent risk hazard and apply the appropriate factors to consider the childrens's risk.

So I hope it would help the Panel and move on with some other discussions. And that's all I have to say at this time.

DR. ROBERTS: Thank you, Dr. Vu, for that clarification. That's an important point. And we need to be careful how we use their terminology in terms of cumulative risk and aggregate to reflect what we really mean.

Our next public presenter is Yvette Lowney on behalf of the American Chemistry Council. Welcome.

MS. LOWNEY: Thank you. You can go to the next slide. In the assessment that --

DR. ROBERTS: Actually, Yvette, you do need to need to identify yourself. Even though I introduced you, you do need to identify yourself for the record.

MS. LOWNEY: I'm Yvette Lowney with Exponent. And I want to talk about some work that I've done on behalf of the American Chemistry Council.

In the assessment that EPA put out recently, they looked at several pathways of exposure to metals from CCA-treated wood. And they included exposes associated with residues on the wood and residues that are in the soils or substrates. And they looked at ingestion and dermal exposure associated with each of these.

I want to talk about is limited specifically to residues and specifically to the ingestion exposures associated with the residues. Next slide.

There's several factors that effect the exposure to residues. The dose is going to be affected by all of these things reflected on the slide and others. I'm only going to talk about the first two issues there.

The first one is dislodgeable concentrations of the residues on hands. This slide says arsenic, but we do have some information about chromium as well. And then transfer of the residues from hands to mouth and different approaches that can be used for doing that. Next slide.

Okay. For looking at the transfer of residues from wood to hands, in the draft EPA assessment, EPA assumes a one-to-one relationship, meaning that whatever has been the measured concentration on wood from wipe data is considered to be the same concentration on hands. And it's expressed as micrograms per unit area, usually 100 square centimeter area. And we think that there's some evidence indicating that only a fraction of what is on hands is -- only a fraction of what is on wood is actually transferred to hands. Next slide.

When we looked through the literature, we found that there are several studies that look at the concentrations of metals and residues on wood. A couple of studies that look at the concentrations of residues on hands, and we found one unpublished report by SCS, which other people have referred to today, that actually has paired data looking at concentrations on wood and concentrations on hands. And those data indicate that hands are much less efficient at removing arsenic and chromium than the wipes are. Next slide.

So I'm presenting the data that are included in the SCS report. Some of the strengths of the study are that it looked at various wood types. The top one listed there was an untreated control. There is one sample in here that is treated with a sealer. They're mostly new lumber, CCA-treated lumber. And what they reported, they did Kimwipes studies of the surface and reported the concentrations on wipes per hundred square centimeters. And then they rubbed the same wood samples with hands, a different area of the wood sample.

And so it was a fairly aggressive approach for rubbing the hands. They would put the hands on the wood, rub the hands forward and backwards, turn the piece of wood on the side and rub the hands forwards and backwards on the surface of the wood again.

So it's a fairly aggressive hand-rubbing sample of the same --

different split of the same piece of wood. Can you move the slide up a little bit so that you can see what's at the bottom.

With the arsenic on or with the data on arsenic across a board, the percentage transferred from the wood to hands is less than a hundred percent. One exception. There is an aged yellow pine sample, and I have the chromium data, which I'll show in a second as well. That value is above a hundred percent. And we think that that's an artifact of how the study was done.

These were boards that were out in the environment. And when they collected the wipe sample, they wiped the top surface. When they collected the hand sample, they wiped the top surface, the side surface, and the bottom surface. And there's some concern that the concentration sort of leached around and collected on the bottom edge of the wood. And that's why the surprising result of more than a hundred percent transfer from the wood to hands.

The next slide presents the chromium data. And, again, very similar results. The average across all of them, except for the control, including the high-aged CCA yellow pine sample for arsenic, was 38 percent; for chromium, it's 28 percent.

The next slide presents these side by side. And you can see that if you take out what we perceived to be an outlier, that the transfer

from wood to hands actually seems to be about one-fourth rather than a one-to-one relationship. Next slide.

So those are the main issues I wanted to present on that topic. What we think is that it's very important that this issue of a transfer efficiency from woods to hands be incorporated into an assessment, any assessment, that is done by EPA.

The data that we have from this one study -- and I understand we're going to leave a copy of this study with the Panel -- indicates that it's about 25-percent, or less than 25-percent, transfer.

I also understand that CPSC is going to be going out and collecting samples from playgrounds. And I think that if the Panel's belief is that we need a more robust data base, it might be appropriate to have CPSC -- not from every site they sample but maybe from a subset of the samples of the sites that they're going to be out sampling -- also collect some hand-wipe data so that we can get more data to base this transfer relation on. Next slide.

So now I want to talk about the second issue which is the transfer of -- the first part was talking about the transfer from wood to hands. And now I want to talk about the transfer from hands to mouth.

There are two ways to approach this. They're reflected in the

risk assessments for CCA that have been done to date. One is to use what I call a behavioral approach, where you look at the behavioral data and try to estimate how many times kids touch their mouth, what's the surface area of the hand that actually goes into their mouth. And that way you can calculate transfer from hands to mouth.

The other approach that can be used is to use an empirical data base. For example, what we know about soil ingestion, which is a fairly strong empirical data base, and use the information in that to calculate what the transfer from hands to mouth is. Next slide.

In the risk managements that I've reviewed over the last couple of decades, what I have seen is that, when a behavioral approach was used to estimate soil ingestion, the values are all over the place. It depends on what you assume, how many contacts, what the surface area is, and it's highly variable.

As soil ingestion studies have become stronger, the empirical data base has become more developed. What we're seeing is that the soil ingestion rates that are predicted by those studies are more consistent and tend to be headed to lower values. Next slide.

So this slide presents a summary of what was done in the EPA assessment. They used a behavioral approach. They estimated for CTE, for the central tendency estimate, that 9.5 contacts per hour

were made from hands to mouth, for their high estimate, 20 contacts per hour. With each of those contacts, they assumed a 20 square centimeter of surface was inserted into the mouth, and that one to three hours was spent on playground equipment.

And then they also incorporated a 50-percent removal. And that's from the hand once it's in the mouth, removal from hands to saliva.

When you put all of this together and calculate a surface area, it yields 95 to 600 square centimeters of surface area involved in this hand-to-mouth contact. Next slide.

The behavioral approach has a very intuitive appeal. I can say that because, when I did an assessment of CCA-treated wood over the course of the summer, I thought, How does this happen does? Gosh, kids get it on their hands; they put their hands in the mouth; it gets transferred from their mouth. And I did the exact same approach.

We used input values very similar to what EPA used, although we adjusted the number of contacts per hour downwards by a factor of three. And that's from a reanalysis of the Zartarian data that EPA also discusses, where she looked not only at hand-to-mouth contact, but actually insertions of skin into the mouth. And she estimated that approximately one-third or less of hand-to-mouth contacts involved

insertions of skin into the mouth. Next slide.

So the problem with this approach that we've been thinking about more over the course of the summer is that the estimates of exposure that you get when you use the behavioral approach don't really square with what we know about soil ingestion. Next slide.

So these are numbers that we used in our assessment: 3.2 contacts of hands to mouth per hour, 20 square centimeters of contact.

Now, Roels looked at -- he was studying exposures of young children to lead. And from his 1980 study, you can calculate a hand loading of soil of .74 milligrams per square centimeter.

So we took that value and plugged it in with the rest of the assumptions that we were using in our assessment. Soil ingestion is assumed to occur during all waking hours, so we multiplied it by 12 hours per day. And we came up with this value of 568 milligrams per day of soil ingestion. That number is not consistent with the current literature. The most recent study by Stanek and Calabrese suggest that the mean soil ingestion rate is 31 mgs. per day. And median soil ingestion rates are lower than that, around 17. Next slide.

So if you take the EPA's assumptions and use them to calculate soil ingestion by incorporating the Roels hand-loading data, you

would estimate that 70 milligrams of soil are ingested in one hour. And that's the CTE estimate. For the EPA upper end estimate, it would be that 444 milligrams of soil would be ingested over the course of three hours.

If you extrapolated their CTE estimate to 12 waking hours, it would 844 milligrams of soil per day, and the upper end would be nearly 2,000 milligrams per day. And this is not consistent, as I said, with the recent soil ingestion data suggesting that daily soil ingestion rate is around 17 to 31 mgs. per day. Next slide.

So instead, if you start with the empirical data base on soil ingestion and take it and back calculate the values for the surface area that must be inserted into the mouth and be contributing to soil ingestion, what you find is that it appears that about 23 to 42 square centimeters of hand surface area contribute to soil ingestion exposures. And that's on full day exposure basis. Next slide.

So here's a summary of some of the recent assessment. The 23 to 42 square centimeters is the value I just explained. Gradient, in their assessment, uses a very similar approach. They used a hand-loads per day and information on the surface area size of young children hands and came up with an estimate of 49 square centimeters per day. I suspect that the difference between the 42 from Exponent

and 49 from Gradient is actually a different daily soil ingestion rate. They were using a previous Calabrese value.

Dr. Roberts used a similar approach and came up with a estimate of 70 square centimeters per day. And, again, those there full-day values.

With the EPA assumptions, if you put them all together and calculated them, you would come up with 90 square centimeters of exposure in one hour for the central tendency estimate and 600 square centimeters in three hours for their upper-end estimate. So you can see that there's a fairly large discrepancy between these approaches.

Okay. This is just a graphic that presents a summary of what I've been talking about. The EPA central tendency estimates would predict over a 12-hour day 844 milligrams of soil ingestion. The central tendency estimate is nearly 2,000. And those values just don't square with what EPA believes from standard soil ingestion assumptions or what the new Stanek and Calabrese data are suggesting. Okay. Next slide.

So conclusions are that using the behavioral data from observational studies will result in an overestimate of the contact rate and ingestion of residues; and that we believe that it's really important ground truth the ingestion assumptions against the

empirical data base on soil ingestion. Thank you.

DR. ROBERTS: Are there any questions? Yes, Dr. Freeman.

DR. FREEMAN: On the Calabrese data, which of his tracers were you using as standard?

MS. LOWNEY: You know, I would need to go back and review the study. I don't recall which one.

DR. FREEMAN: As I recall, there was a great deal of variability depending on whether you were using aluminum or whatever.

MS. LOWNEY: Yeah. The values that we reported here were his best estimates for long-term average ingestion rates.

DR. ROBERTS: Dr. Ginsberg.

DR. GINSBERG: The assumptions of how much soil is ingested as a result of your -- not the behavioral, what did you call it, the other one?

MS. LOWNEY: Empirical.

DR. GINSBERG: Empirical. Right. Regarding that, you have an assumption of an adherence factor of what Roels, et al., 1980, were describing as something like .74. And my understanding of that European study is that those kids were playing in soil and had hands that were fairly dirty, and that we can assume that that's reasonable to

represent that scenario.

But for someone playing on a Playscape, especially given what you were just saying in the first part of your presentation, that the transfer efficiency from relative to the swipe on the hand isn't very high.

I'd just like to hear your comment on how much soil do you think is adhering -- not soil -- dislodgeable material is adhering to a hand relative to what Roels was describing given these kids aren't really playing in dirt. They're swiping their hand across a deck which may not be quite as dirty a situation. And that, you know, the Exposure Factors Handbook is using numbers around the .2 as the central tendency now for children. So, you know, the .74 number just jumped out at me as using it in that scenario.

MS. LOWNEY: The Roels's values collected at the end of the day from children who have been playing at school. A confusing part of this is that I'm not actually saying that I think that .74 milligrams per square centimeter of residues is loaded onto hands. What I'm saying is that we can use that value for soil ingestion to derive a surface area that must be contributing to exposures.

DR. GINSBERG: Based upon 31 milligrams a day of soil ingestion. That's a different scenario.

MS. LOWNEY: Right. What I'm trying to do is sort of clump all of my apples and derive a value and then apply it to oranges. But I think the methodology is accurate. Because somebody earlier was saying, well, it's an awfully unusual unit to express something as square centimeters of contact. But that is the surface area of the child's hand that appears to be contributing to exposures. It's not that I think that the soil-loading rate is relevant to residue loading rates.

DR. GINSBERG: You'll get different numbers if you use a lower dislodgeable loading rate onto the hand. And, you know, the point you're making about the over estimate on EPAs assumptions about how much soil ingestion would necessarily result from your forecast is going to be dependent upon that hand-loading rate. So it's just important to keep that in mind.

MS. LOWNEY: Right. Those hand-loading data are not inconsistent with research that's been done by Dr. Kissel, where it was adult intentionally loading soil to their hands. And if you assume that it all loaded onto the palm surface, those values would be similar.

DR. ROBERTS: Just as a follow-up to Dr. Ginsberg's comments. The thing I was struck by was this was based on 12 hours of -- your comparisons were based on 12 hours of continuous hand-to-mouth activity. Had you picked some different assumptions

for the adherence factor and the duration of exposure in hand-mouth activity, it might not be as large as the numbers that you've calculated. I'll say that. And I'll turn it over to Dr. McDonald is next and then Dr. Kosnett.

DR. MCDONALD: Peter McDonald. Just a comment on the removal of the outlier in the SCS 1998 wood-to-hand data. You had a ratio of 153 percent. So you argued that the hand had been biased towards picking up more than the comparable measurement of the amount on the wood. So you discarded that. But, of course, remember that if things had happened the other way around, you'd get a low outlier and you probably wouldn't have flagged that and wouldn't have removed it. So removing only the higher outliers will bias the data.

So my question is: How much replication was there in that data set that would let you get some idea of the reliability of those figures, and whether, say, the 153 percent was plus or minus 20 or plus or minus 1 or whatever?

MS. LOWNEY: Right. As I said, we'll leave a copy of that study with you.

There were five volunteers for each wood sample, and each one did a right hand and left hand. So there is some replication in there

that we can go and look at. If that value of greater than a hundred percent were close to any of the other values, I might not have been inclined to dismiss it as an outlier. But it is so inconsistent with all of the other data, that it just really jumped off the page at me as being an outlier.

DR. MCDONALD: Surely, that would be a case for somebody that ought to be repeating the trial just to confirm what's really going on.

MS. LOWNEY: And therein is the reason why I'm recommending that a neutral body CPSC might want to pursue a similar study.

DR. ROBERTS: Thank you. Dr. Kosnett.

DR. KOSNETT: What do you think is the best study on hand-loading of children? I mean, what would you recommend that the Committee review and consider as the definitive or best study?

MS. LOWNEY: I think that the data base on hand-loading is very limited and that the methodologies that they used are very disparate and that it's very difficult to pick one study. You know, there's either a small or a huge disparity within the data base.

Actually, that is why in our approach we decided that it would be better to establish a wood-to-hand transfer relation because the

data base on wood concentration is more robust and there is more data going to be collected. So then we have a robust data base that we can then apply this transfer rate to.

And, also, our thought was that that encourages further data collection. I think it's easier to suggest that municipalities or an agency go out and collect samples from playgrounds than it is to ask them to conduct studies with humans.

DR. KOSNETT: And is the hand, the wood-to-hand transfer study, the one that you would like us to consider, the SCS study?

MS. LOWNEY: The SCS study is the only study that we found that had paired data of both wood-loading concentration and hand-loading concentration. So that's why we selected that to establish the transfer ratio.

DR. KOSNETT: And you've supplied that to the Committee.

MS. LOWNEY: Yes, we will. I'm having a copy sent over.

DR. ROBERTS: Dr. Smith, then Dr. Freeman, Dr. Wargo, Dr. Matsumura, and Dr. Kissel.

DR. SMITH: I was trying to figure out if Dr. Freeman had discovered that this was a new way to get ourselves called.

I've got just a couple of questions, first a simple one. In that first data slide that you put up showing data for wipes versus data for

hands, the data for wipes of micrograms per centimeter squared, I assume that's centimeter squared of surface area that was wiped; is that correct?

MS. LOWNEY: Correct. We wiped a hundred --

DR. SMITH: Or is it the surface area of a piece of wood and you may have repeatedly rubbed it?

MS. LOWNEY: No, I'm sorry. They wiped a hundred square centimeter surface area of wood, and that was the total residue for that area that they wiped.

DR. SMITH: And the units for the hand, microgram per centimeter squared, is that for the surface area of the hand because people seem to do it different ways; or is this, again... So what's the units for the hand?

MS. LOWNEY: I understand. The data they reported were micrograms per hand. So it was a hand-loading study. They also did traces of the hand and calculated the surface area of each hand. So from that you can calculate hand-loading per hundred square centimeters of hand. And that's what I presented, and that's why.

DR. SMITH: So we have two different types of measurement. One is micrograms per centimeters squared of wood surface which may have been wiped multiple times in different directions. And then

the other unit is a microgram normalized to centimeter squared of hand; is that correct?

MS. LOWNEY: I believe so. One is microgram per hundred squared centimeters of wood. The other is micrograms per hundred square centimeters of hand.

DR. SMITH: Of hand. Thank you.

The next question is -- sort of just help me think through this a minute from an intuitive point of view.

You've got an estimate of soil ingestion that's a central tendency measure. So this is the sort of typical kid's soil ingestion rate. Why is it that we should think that soil ingestion data may provide us a better estimate of hand-loads of childrens' behavior per day for the pressure-treated wood scenario?

And the reason why I'm asking this is I'm trying imagine a young kid who's got visible dirt on their hand and the frequency that that hand's is going to go into their mouth versus a child that's having contact with pressure-treated wood and there's nothing apparent on the hand or very little apparent on the hand.

I could possibly see making an argument that the approach you're taking may represent a good lower bound for us to keep in mind. But it's not as clear to me from an intuitive point of view that it

would necessarily be reflective of the same behavior. So I was wondering if you would talk to us about what your thoughts are on that.

MS. LOWNEY: What I like about using what I'm calling the "empirical approach" is that -- in the calculation I showed at the very beginning, just the descriptive calculation, where there were these parameters that go into estimating exposures. And, you know, within that, then there are all these parameters that go into evaluating. If you're using the behavioral approach, there are all these parameters that go into calculating what the hand-to-mouth transfer residues is.

And we don't know -- Kevin, you've done a lot of assessments -- we don't have hard numbers for any of those. They're based on observational studies; and, gosh, do they really put their hand to their mouth that often? Do they touch the wood and reload the residue onto their hand before they touch their mouth again? Or do they touch their mouth twice in a row before they reload again?

There are all those issues that are very difficult to answer. I'm tempted to say unanswerable, but certainly very difficult to answer. And those come together as a factor that we're missing in the calculation when we've used the behavioral approach.

If you use the soil ingestion rate and calculate what -- if you use

the empirical approach that I'm talking about, you come up the surface area of the hand that appears to be transferring anything from the hand to the mouth, whether it's residues or soil or skin cells.

DR. SMITH: May I interrupt? I understand the logic of it, so I don't have a problem with logic of it. I'm trying to ask a question of why is it that we should think the soil ingestion behavior of a hand probably having visible dirt on that that's going to reflect that a child is going to put that in their mouth as often as a child who is just playing on a pressure-treated structure and there's no visible dirt on their hand, especially as we start to deal with two-, three-, four-, and five-year-olds that may have that behavior.

MS. LOWNEY: Right. The reason that I'm making that link is that my understanding of soil ingestion is that it comes primarily from inadvertent hand-to-mouth contact. And our concern about exposures by young children to residues that they come into contact is that the pathway also involves inadvertent transfer from hands to mouth. And so it's a parallel exposure pathway. And, therefore, the data can be used. The data from the soil ingestion can be used to assess exposures to residue.

DR. SMITH: And one last brief one. This is to follow-up on a comment that a couple of the other SAP members have brought up, but

let me ask it from a slightly different direction.

Ideally, what we would have, if we were going to do your approach, would be with the same data set we would have paired data where we have the loading onto the hand as well as some sort of estimate of ingestion rate. And we don't have that.

What we have is an ingestion rate from one study of young kids, I believe. And then we have a soil-loading estimate or the adherence-factor estimate from a study of older kids. And one would expect some sort of correlation between these that, the higher the loading onto the hand, probably the higher the soil ingestion rates. So you don't have that.

So I would just ask you to take a close look at the values again that you're using for soil adherence and make sure that we're looking at similar measures; we're not using a high end of one and a low end of the other. Because it strikes me, again, as has been mentioned by Dr. Ginsberg and others, that the .74, I believe, was a higher-end estimate, and so you may be biasing a result. Because, again, ideally, what you'd like is paired data; and you do not have that.

DR. ROBERTS: Dr. Freeman.

DR. FREEMAN: I was thinking back to the way you were calculating things. And one of the things that you assumed was that

this three-and-a-half-times per hour occurred over 12 hours of the day. There's no literature that I know of, including INHAPS and NEXUS and other things, that suggest that children are in contact 12 hours a day so that you're overly inflating the potential for having a soil loading on the hands. That if you actually use what EPA is intending to use, which is one hour of contact or three hours of contact, that you might get very different numbers.

MS. LOWNEY: Right. My understanding of soil ingestion is that there are contributions from outdoor sources and contributions from indoor sources and that it is believed to continue over the entire waking period of a child. That's why I conducted those calculations that way.

If you go through the slides, I calculate a variety of different ways specifically for this. I did calculate the surface area of transfer using the EPA assumptions and other assumptions. So I think if you use all of the data that I've just presented together, you can address that question.

DR. FREEMAN: The one issue is that because you used Roels outdoor after a day of play loading on the hand you're getting something very different from the types of loadings you get on kids when they're playing in house dust, which is more like .03 milligrams

per centimeters squared. So it's much smaller.

And, yes, a good portion of that is from outdoor soils that have come into the house by one route or another. But the loadings you're getting because of the characteristic of house dust is much less.

DR. ROBERTS: Okay. Dr. Wargo, Dr. Matsumura, and if he's still interested, Dr. Kissel

DR. WARGO: I'll be brief. Were you here for the presentation that described the simulation across the different sources, then Environmental Working Group presentation?

MS. LOWNEY: Yes.

DR. WARGO: I'm curious about that. You seemed to be suggesting that certain data be used that describes central tendencies or mean levels. And I've also seen the Agency and some of your documents presenting mean concentrations from different sources.

And the question is about kind of your thinking about the appropriateness of the method that should be applied here and whether or not it makes sense to use the full distribution of data points that we would have and then sample from those and then aggregate across sources as away of coming to some view or some projection about how one individual might accumulate the exposure and then move on to the next individual. It seems that that's a richer way to deal with some of

the uncertainty that's involved in the data sets that you're using.

MS. LOWNEY: Right. My area of expertise is not probabilistic assessment, so I feel that I can't really discuss that. What I can say is that the reason we were using means and medians was because we're interested -- the exposure period that we're looking at with this scenario is fairly long and in which case there would be a tendency towards the means over time. So using some sort of central tendency for a long period of exposure would be appropriate, I believe.

DR. WARGO: I think now's not the time to do it, but we should have a conversation about that.

DR. ROBERTS: And it may come up. Well, we'll certainly be discussing that. That is one of the questions posed to us by the Agency as whether or not a probabilistic risk assessment would be the way to go on this. Dr. Matsumura.

DR. MATSUMURA: I will be quick because my questions have been asked by Andrew. I have just a quick question.

When you are considering the transfer from the hand to mouth, you are include studying a hundred percent going in or you are considering some other factors?

MS. LOWNEY: Oh, good question. When we first looked at this and used a behavioral approach to assess exposures, we did

assume that there would be a hundred percent transfer. By using the empirical approach, I don't need to estimate what that value is because it's done for me. What the value expressed is the area of hand that appears to contribute to exposures. And that will incorporate issues associated with how much is off-loaded into the mouth.

DR. ROBERTS: Dr. Kissel.

DR. KISSEL: Yeah, a clarification.

DR. ROBERTS: I'm sorry. You're going to have to use the microphone.

DR. KISSEL: On that last point, in fact, I guess that was the clarifying question I wanted to ask, also. When you say 23 to 42 square centimeters, you mean 23 to 42 square centimeters that are completely extracted and from the content is ingested because it has to be that.

MS. LOWNEY: Right. That's right

DR. KISSEL: So in fact, the assumption -- the actual amount of skin that goes into the mouth could be much larger than that, but it's equivalent to 23 to 42 by your calculation from which you completely extract the dirt and ingest it.

MS. LOWNEY: Thank you for clarifying that.

DR. KISSEL: On the wood-contact test, a couple of questions.

One is: Was extraction of the hands tested? Because you can digest a wipe, but I can't digest somebody's hand. So you don't know that you got complete removal of what was on the hand when you're comparing those two.

MS. LOWNEY: The hands were washed before wiping, and then they were washed after the contact. So it was a washing of the hands.

DR. KISSEL: Okay.

MS. LOWNEY: It wasn't a wiping of the hands.

DR. KISSEL: Yeah, but was an attempt made to do a mass balance on a hand, you know, load the hand with something that you knew was there and then remove the stuff and see if you've got the mass balance that you think you did?

MS. LOWNEY: No, I don't believe that was part of the study.

DR. KISSEL: It's a hundred square centimeters of wood was wiped. But how did they control that you only wiped a hundred square centimeters of wood with the hand?

MS. LOWNEY: Actually, the surface area of the wood that was wiped with the hand was far in excess of a hundred square centimeters. So it is -- they put their hands on the wood and moved them forward four inches and back a series of times and then forward

and back. So it is not a -- the data can be expressed as loading per area of wood that was contacted.

My concern about expressing it that way is that, then, what would need to go into a risk assessment is what is the area of wood that a young child contacts when they're on a play structure. And we certainly don't know the answer to that.

Some of the data that were collected by the Maine Bureau of Health looked at hand-loading. And what I see in those data is that they did a variety of things. And it's very instructive data because they would wipe one surface and measure the hand-loading. They would wipe two surfaces and measure the hand loading. They would rewipe the same surface and measure the hand-loading.

And what it looks like is that the transfer from woods to hands -- saturable is a word that I might use -- there's a certain amount that gets on your hand and then no more gets on your hand. And to the extent that that's true, it makes the risk assessment methodology that we need to use much simpler.

We will certainly have lots of discussion about behavior if we get into how much surface area of wood a young child contacts.

DR. KISSEL: Okay. But what that does raise, though, is that getting a number on the hand which is number higher than the number

you got off the wipe is not at all unreasonable then. If I put my finger down on a surface which has a known loading and I just have one static press and then pull it off, I don't expect to get a hundred-percent transfer. I would be stunned if I got a hundred-precent transfer unless it's peanut butter and jelly on glass or something like that.

If I take my finger and swipe it down a larger area so that the area of the finger that touches is much smaller than the area of the surface, then it's easy to get a loading higher than the loading that you started with.

MS. LOWNEY: Conceptually, I agree with that that you could get a loading that was high; but I don't believe that these data supported that.

DR. KISSEL: Okay. And the last comment has to do with the getting to the 23 to 42, which other people have already brought up. I do think the Roels number is probably too high for just a normal situation. Plus the Roels data, it's not a primary measurement of soil loading on skin; it was a measurement of lead which was then converted to soil which makes the uncertainty bounds on those particular numbers larger than maybe on other numbers.

And the ingestion numbers, I like what you're trying to do. I

like trying to close the circle and make things make sense. I think we ought to be doing more of that. But I think the Calabrese's numbers keep going down. And there are data sets out there that they have to match up with, which they don't match up with, if they keep going down.

I've got some urinary arsenics in kinds from the Ruston-Tacoma Smelter area, and I can't explain those body burdens if the soil ingestion numbers are as low as Calabrese says they are now. There's no reason to for me to believe that a soil ingestion study of the type he does, where you look for tracers coming out of the body and then you try to back calculate based on what's in the environment, is inherently better than a real-world experiment where you have kids living in a contaminated area and some of those things are showing up in them.

Those are tracer experiments, also. And I'm not really happy with where those numbers are going at least for some of the time. Because, you know it may be a summertime thing. His numbers may be okay for annual averages. But I think certainly there are -- I can cite some cases where I can't explained observed body burdens if soil ingestion numbers are down there around 10 milligrams a day.

MS. LOWNEY: Right. Our point is that -- the way we looked at this, with the empirical data that we put our hands on and did the

calculations, suggest that the behavioral approach doesn't square with the empirical data. If there are better empirical data that you want to use to substantiate the value that's used in the risk assessment, I think that would not be inappropriate.

DR. ROBERTS: Dr. Hopenhayn-Rich, and then Dr. Ginsberg.

DR. HOPENHAYN-RICH: My question was already asked and answered.

DR. ROBERTS: Okay. Thank you very much. Dr. Ginsberg.

DR. GINSBERG: Regarding the wipe-to-hand transfer efficiency, I just want to clarify that the SCS study used dry hands; is that right?

MS. LOWNEY: They washed the hands before they were rubbed on the wood. They were dried but probably damp.

DR. GINSBERG: Probably damp.

DR. ROBERTS: Any other questions? If you really want to ask this last question, go ahead, Dr. Smith.

DR. SMITH: If there is any chance that they could have been damp, that could easily explain a very large difference in hand-loading.

DR. ROBERTS: Thank you very much for your presentation and patiently answering all of our questions. Our next speaker is Dr.

Barbara Beck.

Welcome. Could you introduce yourself for the Panel.

DR. BECK: Yes. Thank you, Steve. My name is Barbara Beck. I'm a toxicologists and risk assessor at Gradient Corporation. And over the past few months, we've been involved in performing risk assessments for CCA-treated wood.

I'm only going to briefly describe the risk assessments to you. My aim is really to provide some input regarding the issues raised for the Panel and alternate recommendations for approaches.

We provided one, what we called a focused risk assessments, to EPA and CPSC in July which involved a limited number of exposure pathways. Basically, no sensitivity analysis, little analysis of arsenic toxicology.

Since then, we've preformed a more comprehensive assessment, which I believe the Panel has, looking at CCA-treated wood. We call it more comprehensive because we looked at both playground exposures as well as residential exposures. And although I understand the focus here is playground, we did look at residential exposures which, of course, would have a higher frequency of exposure either to a deck or to a play structure.

What I have presented here is just a snapshot of some of our

results. This is for the child ages 2 to 6 at a playground, although we did also look ages 7 through 12, recognizing that older children play, of course, at playgrounds as well. And we looked at two different types of treated wood in our analysis that were derived from the SCS study. We looked at the type of treated wood that had the highest dislodgeable arsenic on the surface.

And what I present here is the wood type which is the most common treated-wood type on the market. It's plain old southern pine treated with CCA. It is not sealed. It represents about 86 percent of the market of CCA-treated lumber in the U.S. And we looked at both and our mean estimate of risk as well as the CTE estimate of risk.

I just present the cancer numbers here. You can see that for soil, we look the at three pathways suggested by EPA. I'll also confirm that inhalation turns out to be negligible. And for dislodgeable, we looked at ingestion and dermal exposure.

And the results of the risk assessment for this particular element is that the cancer risks from dislodgeable and soil arsenic in the playground setting all fall within EPA's acceptable risk range. The highest value there is 1.5 times 10 to the minus 6. This is, again, for regular southern pine. The value is several-fold higher for southern pine with factory treated water repellant.

And then if you look at our residential risks, the values are about three- or fourfold higher; although they all fall within the EPA's permissible risk limits of one in a million to one in ten thousand. Next slide.

This just lists the issues that I'm going to cover. They correspond to the questions that were posed to the Panel by EPA. There are other issues that are being addressed, for example, by Yvette Lowney. Some will be addressed by John Dutalla and Joyce Suji. Bioavailability is Issue 2. The Key Exposure Parameters are Issue 7. The Suitability of the Data for Probabilistic or Monte Carlo Analysis is Issue 8. Next slide.

Issue 11 is what is the appropriate exposure point concentrations to be using for both dislodgeable and soil metals. And then 12, 13, and 15 are the last three, which I will only touch on. I plan to focus more on the previous issues. Next slide.

Bioavailability is going to be a very important parameter in this assessment. It's always an important issue in conducting risk assessments for metals, metals in soil, as well as other media.

Just to define, again, what is bioavailability. What you need for risk assessments, of course, is relative bioavailability. And in this case, what is the bioavailability of CCA arsenic in soil or

dislodgeable versus arsenic in water. And the reason is that's the form of arsenic that forms a basis for our toxicity study, whether it's Musumder or Tseng or Khan. Those are all drink water studies.

And you'll see, because of the need for that adjustment, some of the recommendations that I'll be presenting differ somewhat from those of Dr. Aposhian in that in that he presented absolute bioavailability estimates.

The value that's been recommended is 25 percent based on a synthesis of the work of Dr. Freeman and Dr. Roberts in particular. We believe that it may be more appropriate to consider soils that have actually been affected by CCA at a treatment site.

I recognize that this is not the same exactly as what might be present under a play structure where you may have different processes involved in releasing that material. But it seems that as a first approximation, it's not an unreasonable way to start. This, as you know, is based on studies with primates fed soil from a CCA-treatment site.

Now, I believe that this also can be used in terms of what we think for the dermal uptake value that the information from oral bioavailability studies are basically a reflection of the bioaccessability. In other words, how readily solubilized is arsenic or

lead in the GI tract. And then what is solubilized in the GI tract, the bioaccessable form, then absorbed into the body.

That bioaccessability factor is not an unreasonable way to think about what to be doing for dermal uptake. And, in fact, if one thinks about it, one may think that the GI tract would be a more aggressive solubilizing medium than the sweat on the surface of the skin. Next slide.

Now, specifically for dermal uptake, EPA is recommending 6 percent based on the study of Wester involving soluble arsenic. In contrast, the default value in the exposure -- sorry. Not the exposure factor -- in other EPA guidance for arsenic is 3 percent based on the same studies, looking in particular at soil.

We believe that this is a reasonable way to start. It's perhaps conservative. These are studies in which the animals had freshly -- soil was freshly -- arsenic was freshly added to soil. It was placed on the skin of the animal. It was occluded. So it was conditions that would yield a higher uptake than soil that might be aged in the environment and have opportunity to bind to soil. And the use of an occlusion patch also would reflect conditions that would facilitate uptake.

So we believe that one needs to consider using that 3 percent as

a starting point and then thinking about how to adjust that for a reduced bioavailability from soil as a consequence of it ageing in the environment or other factors.

For example here, one might say, well, you could apply 16 percent by that 3-percent value. There may be other ways to consider this, but we do believe that there needs to be some consideration of modification to that 3-percent starting point. Next slide, please.

Now, the dislodgeable is really an important element. I'll get into later how our analysis indicated that overall, in terms of overall risk, ingestion of dislodgeable arsenic is really a driving parameter or driving pathway as far as risk goes. So in terms of collecting additional information, this is something that's important to think about.

When we started our analysis, we did not have the benefit of Dr. Aposhian's study. There was a study out there from Peoples and another from Peoples and Parker, dogs being fed ground up CCA-treated sawdust. I will admit fully this study is old. It does not have a large number of animals. And nonetheless, it indicated a relative bioavailability which we calculated ourselves of 47 percent.

We felt comfortable starting off with that number even though it was based on a limited number of animals because we had also had

leaching studies in which blocks of wood of various sizes were leached under acid conditions, one normal HCL for different periods of time. And the arsenic that came out under those conditions, which are somewhat similar to the stomach part of the gastrointestinal tract, were 17 to 44 percent. We at least felt we were in the right ballpark.

We now have the study from Dr. Aposhian with the value of 11 percent for an absolute bioavailability estimate. We believe that, in order to use that risk assessment, we need to consider what is the absolute bioavailability of soluble arsenic in water as Dr. Aposhian presented.

There are a number of estimates out in the field for that value. So we said, well, what if it's really a hundred percent, then the oral dislodgeable value may be on the order of 10, 11 percent. If the absolute bioavailability of soluble arsenic in water in the hamster is as low as 50 percent, then that would increase that relative bioavailability estimate up to 20 percent. So it kind of gives you a ballpark estimate of possible values to consider.

Again, we believe that this could be applied to the dermal uptake that there might be some adjustment to that 3-percent value. Next slide.

Now, soil in -- sorry. I'm skipping ahead of myself.

The skin surface area for dermal contact with dislodgeable in soil and metals is another important parameter for the assessment. We believe that it's important to consider how children come into contact; how their skin surfaces come into contact with the wood surface and with the soil; and that's it's also important to consider it in terms of typical exposure conditions under longer term exposure, say six months. So it's in our assessment for the dermal pathway for soil. We actually choose a value that was higher than what EPA chose by a factor of about two.

For our dislodgeable assessment, we did not consider skin surface area other than the hands. That's something that we are rethinking. I think that it still is reasonable to consider that that's going to be limited primarily to the hands, given that it is a flat surface that kids are contacting; but that we might want to consider other body-part contacts with a reduced frequency. And I'll get into some assumptions as to how we might be able to address that. Next slide.

Soil Ingestion Rates. There is some debate about soil ingestion rates in the literature. I think we know that every few years Dr. Calabrese looks at his data a different way and we have slightly different distributions.

What we've done has been to look at his studies. And we think that his estimate for soil ingestion rate, where he uses what's called his best tracer methodology, is a good approach. He looks at a number of different tracers and selects tracers that have the lowest food contribution to body burden. So you don't have problems with signal-to-noise ratios. He also looks for consistency among tracers, and then chose the median value of, I believe, it was four tracers.

So in answer to an earlier question, there are a number of tracers that are involved in his best tracer estimate; but they give a fairly consistent number, and they're the ones that are best in terms of having relatively low-food contributions which can really give very uncertain estimates.

We chose the results from his Anaconda study. I'm sorry. We chose the results from the Amhurst study. The results from the Anaconda study are actually somewhat lower than the values here where we wonder whether it may have to deal with issues regarding particle size of the soil that was measured for the ingestions studies, that it may be important to look at tracers levels in smaller particle size that adhere to kids's hands. And if your tracers vary as a function of particle size, that can introduce some uncertainty into your assessment.

Anyway, his median value is the 50th percentile child is 36 milligrams. We believe that's a reasonable central tendency estimate. And then the 95th percentile value -- and this is averaged over ages two to six -- which are the ages that we looked at in the risk assessment. It's a hundred milligrams per day.

Now, there are some estimates that are higher values as high as 400 milligrams. That is based on a brief study period, and we feel that is not representative of usual intakes especially if you're looking at exposures averaged over several months. Next slide.

Now, as Yvette Lowney described, the hand-transfer efficiency is a really important parameter to consider. And she provided a lot of insight as to why this methodology gives results that we believe are consistent in what's measured in the real world. I'll get into this later, but to address some of the questions that have been asked here about what's the appropriate loading to be using for soils.

We did use the Roels study, what's an appropriate soil ingestion rate to be using in this analysis. Those are the two key parameters.

We did do a sensitivity analysis to understand had we chosen -- impact on our hand-transfer efficiency. And there are values that could increase the hand-transfer efficiency by several-fold. There are values that could decrease it by several-fold. So we believe that what

we have is a reasonable estimate of a high-end value, and I'll get into some of the details on that later.

As far as the hand-loading studies, you know, the Roels studies, I do agree are surprisingly high. Nonetheless, I think, that from the studies that are out there, it's clear that what's on the hands for soil tends to be higher than what's on the other parts of the body.

We thought that the Roels study had an advantage in that it was kids doing whatever they do and just measuring them at the end of the day rather than looking at specific activity patterns. But one could certainly consider other parameters; and you'll see that we did look at that to some degree in our sensitivity analysis, which is both in the comments that I have as well as the risk assessment itself. The next slide, please.

Another key point is exposure frequency. How many days per year does a child come into contact with a play structure, for how long does that contact occur. And we used data in which it was from the Exposure Factors Handbook in which there are estimates of how often kids go to parks, how long kids spend at parks.

There are also estimates in the Expose Factors Handbook for how long kids are outside at their residence. There's even estimates for adults when they're outside at their residence, how much of the

time are they mowing the grass and doing activities that wouldn't bring them into contact with decks.

So there's a fair amount of literature out there regarding exposure frequency. Unfortunately, sometimes you get adult data from one study or park data from another study. They're not all necessarily from the same study. Sometimes they're a one-day recall diary. Sometimes they're yearly recall estimates. So there's a number of elements to consider.

When we did this, we concluded that 130-days-per-year was not an unreasonable central tendency estimate; but that we did need to consider some adjustment for exposure time. And the reason is that when we think about hand-transfer efficiency, which is really one of the critical factors in looking at dislodgeable, that's based on soil ingestion.

And from what we can tell, soil ingestion occurs over a whole day. And the reason I say this is based on studies from Dr. Calabrese. If you look at how much soil a child ingests that is from outdoor soil versus how much is from house dust that contains soil. And you can do that because there are tracers in house dust that differ from some of the outside tracers. And so it allows you to estimate how much soil is ingested inside as soil, how much represents soil that is tracked into

the house that is ingested inside as house dust. And it's about a 50-50 split, which says to me that it's reasonable to estimate that that process goes on over the course of a day.

Since that process goes on over the course of a day, if a child is ingesting dislodgeable arsenic over 3 hours, then we need to have some adjustment for the fact that we're using a soil ingestion rate that's over a 12-hour day; and in that case, you will need a adjustment of one-forth.

So in this example here, I'm assuming, if you're at a playground one hour a day for 365 days a year, that's equivalent to 30 days of exposure. Because if you were ingesting soil for only one hour a day and not at all for other hours of the day, that would reduce the soil ingestion by a factor of over 10. Next slide, please.

Now, the soil adherence factor is another important parameter for several reasons; and I think it's important to consider it on several levels. EPA is proposing a value of 1.45 as central tendency for arms, hands, and legs. This is based on studies involving, potting soil, involving volunteers in which people place their hands on potting soil, and the loadings on hands were measured.

We believe that one really needs to think about the adherence factor in terms of body parts. And the adherence factor does vary

according to different body parts. We actually came up with an alternate value for Roels, which was higher than the one presented by Yvette Lowney, of 1.1. We went back to the original data and reanalyzed it for different age groups and reaveraged it.

But overall, we wound up using for soil a weighted average of .34 milligrams per centimeter squared assuming that other parts had .24 milligrams of soil per centimeter squared. And I believe that a similar adjustment could be considered for the dislodgeable arsenic. I guess I don't have a slide for that.

Here you can see we have a ratio of about 5 to 1 for hand to other body parts. I don't know -- we don't know what the reason for that is. Presumably, it is that there is just less contact with those other body parts than the hands. This may be a way of addressing the dislodgeable dermal contact. One could either think about reducing the contact frequency of other body parts versus the hands, or one could think about using a different dislodgeable fraction on other body parts versus the hands and assuming that what you're looking at there is a reflection of differences in contact frequency. So I think that the same concept needs to be considered with respect to dislodgeable and dermal uptake. Next slide.

Probabilistic analysis, a Monte Carlo Assessment, ia certainly

something that is a very important methodology for looking at variability and uncertainty and risk assessments. It is certainly a method that we've used in a number of risk assessments. I'd say it's primarily used in situations where we understand the variability and parameters and we have a good sense of distributions. In other words, parameters which differ among individuals, such as body weight or soil ingestion rate.

It's also important if one does an Monte Carlo simulation not to be mixing up variability which varies among individuals versus uncertainty. The lack of true knowledge which, I think, is one concern I have with lumping various data sets for looking at loadings of arsenic on hands or loading of arsenic on surfaces. You're looking at a combination of variability and uncertainty. And then you wind up with an output that is very difficult to interpret.

One can look at availability and uncertainty in Monte Carlo assessments, and we've done that. But you need to distinguish them. You need to look at variability and then one can layer an uncertainty assessment on that.

Given that our assessment indicated that the most important parameter is really dislodgeable arsenic and ingestion and that some of the key parameters there are hand transfer efficiency and exposure

frequency about which there is a fair amount of uncertainty, I think at this point it's hard to develop good distribution estimates for those parameters; and I think it would be very difficult to perform a Monte Carlo simulation with the data we have.

What we did to address this, which hopefully will address some of the questions that had been raised earlier about the inputs into the hand transfer efficient, for example. We started off by looking at dislodgeable arsenic and ingestion. And we looked at alternate parameters.

So for example, the hand-transfer efficiency assumed 36 milligrams soil ingestion. We looked at what would be the impact if we choose 100 milligrams soil ingestion. We compared our parameters with both 5th percentile values and 95th percentile values, and we looked at our RME parameter in particular. Our aim here was really to assess whether we could say with some confidence that our RME value did represent a high-end value.

And what we learned was that oftentimes our RME value was very similar to a 95th percentile value parameter such as hand-transfer efficiency. We did calculate that you could have used a value as much as fourfold higher. But overall, our RME values on average were a factor of two or a bit less versus a 95th percentile value.

And we felt that since what we're looking for here is an overall reasonably high-end exposure but not something that's implausible, we don't want to use a maximum for each value so that we wind up concatenating maximums and minimums and come up with an improbable estimate at the end. We felt that overall this confirmed our RME value being representative of a high-end exposure. Next slide.

Now, the exposure-point concentration for dislodgeable and soil metals is obviously critical. And it's particularly critical, I think, for the dislodgeable. I think we know pretty well how to collect soil data. I think it's important that when we collect soil data that it be representative of the soil that children are exposed to.

And if we're looking at data around the foot of a deck or play structure, we need to consider the whole area that a child might being exposed to. It's more complicated with respect to dislodgeable.

Now, EPA recommends using a mean value for cancer and a maximum value for noncancer. We would recommend 95 percent over confidence limit on the mean for both. That this is really appropriate for the kind of subchronic, say, six-year exposures that we're looking at.

Now, as far as how one measures dislodgeable metals, there's a

number of studies that are out there. For our assessment, we relied on the SCS study because we felt that it had a methodology that was well described. We felt it reasonably conservative. People were rubbing their hands 10 times on the wood surface. It considered a number of different types of wood treatments.

And I should say that of the wood treatments that it used only one was a truly sealed sample with polyurethane. And that's an important point for consideration by the Panel.

I've been guilty of this myself. When we say sealant, I think it's important that it really be an impervious material that prevents water from going in and it prevents arsenic or metals from going out. There are products on the market which are called stain sealed that are not true sealants. There are products called brighteners, stains. These are not true sealants. When we think about sealants, it's important that it really be precisely defined.

Another factor to consider with respect to dislodgeable metals, which we did not consider in our assessment, is the role of aging. And what this refers to is that the fact that over time the release of dislodgeable metals diminishes with these samples to levels perhaps on the level of 20 percent of what is there at present.

The SCS study did demonstrate the wipe samples. Only one of

the samples in the SCS study, by the way, was aged. In fact, most of the studies out there, many of them did not involve aged wood did show the impact of ageing. The hand wipes interesting did not because they were wiping the bottom of the surface with their hands which includes woods that hasn't had an opportunity to truly age.

I think that the playground study is going to be very important here, that getting a sense of what's really out on the surface of those playgrounds that have been out in the real words in different parts of the U.S. over time is important.

The SCS ageing study that we looked at was Florida aged. But I think it's important to consider other parts of the country. And I would, also, really recommend that there be some consideration given to doing concomitant hand-loading studies at the same time.

Now, I realize you're not going to send 10 volunteers to 25 playgrounds and have them wiping their hands on woods all over the U.S., but that it may be possible to either consider a subset of those play structures or to even take part of those structures back to a laboratory so that one can look at hand-loading in some reproducible and reliable manner.

I think that in general, dry wipes are going to be more directly relevant than wet wipes or wet loadings. The reason is that the wood

does tend to dry out the hand. I think that if there is consideration given to using data from either wet hands or wet wipes that you need to consider that this is not going to be something that occurs a hundred percent of time. That at the very least there needs to be some weighting of wet versus dry samples. Next slide.

As far as soil goes, again, as I mentioned earlier, it's important to look at exposure unit. Not just what's around the base of a structure, but what represent the area to which children are exposed. This is how we look at lead risk assessments. This is how we do risk assessments at superfund sites. We look at the exposure unit.

The ground cover is an important issue as to particularly considering that ground cover may be changed over time and that may be a way to reduce exposure. But it is difficult to assess and quantify exposures of wood chips. We don't have any wood-chip ingestion studies. We saw that there's even tire chips that are used, and we don't have tire-chip ingestion studies.

But I think that at least one could then sieve those samples to a particle size that we know adheres children's hand, and that one could use sieve samples and, as a first approximation, consider some of the same hand-to-mouth transfer activities used for dislodgeable as a way to address the sieved samples. Next slide.

The last three issues I just want to touch on. How do you combine multiple exposure pathways and routes? I think one important thing to consider is we don't want to be double counting. So if the child is doing one activity, say, on a play structure, it may reduce their contact with soil. Or the time that they're at the playground, it's important to consider they're also not in their back yard.

Inhalation exposure, I agree, is not likely to be important. We actually did in our risk assessment a soil erosion model and estimated particulate levels of arsenic. And using EPA's cancer slope factor for inhaled arsenic, we still come up with, at present at least, it indicates greater potency than the ingested form of arsenic. The risks are still very low as far as inhaled soil particles containing CCA-treated materials. Next slide.

What is the effectiveness of coating materials in reducing leaching of metals? I think, first of all, there's an issue as to the necessity of it based on the results of the risk assessment. In any case, I think the results to date are inconclusive. There are some data from CPSC that did not show an impact. There's data that says that if you put polyurethane, at least in the short term, you do see a reduction in release. So I think that this is an area where further research is

needed.

I think one also needs to consider how well these sealants perform in the outside world. That polyurethane treatment is something that, if you did treat your deck that way, would require constant renewal. It's not a treatment that's made for outdoor treatment.

And then conclusions. Just to get back to our risk assessment, we believe that this was a conservative risk assessment on a number of levels.

First of all, I didn't take into account any reduction in exposure for release of dislodgeable over time. We choose bioavailability of about 50 percent for dislodgeable, whereas it now appears it could be between 10 and 20 percent. And then we did do a sensitivity analysis where we looked at alternate assumptions to see if that would have a major impact comparing alternate assumptions, both 5th percentile and 95th percentile with the values that we used. And we believe that we are looking at a high-end exposure here. Thank you very much.

DR. ROBERTS: Thank you. Are there any questions for Dr. Beck regarding her presentation? I see several. Dr. Mushak, then Dr. Bates.

DR. MUSHAK: Two quick questions, Barbara.

The soil bioavailability factor for the CCA site you take at 16.3, which is an adjustment downwards from the Roberts 25. You can't really say whether that adjustment or the difference is due to the CCA residue or whether it's due to soil type. I mean it could just as well be that that particular soil that had that CCA residue happened to show a lower BA. So what you would have to do is look at the same CCA residue in two different soil types at a very minimum.

DR. BECK: I agree that one soil sample is not ideal, and I would certainly like to see additional data. This is what we did as our first approximation. But I agree that it would be useful to have additional soil samples, ideally from under a deck.

DR. MUSHAK: Right. Could you comment on the potential mobility of dust under playground equipment as a function of aridity, that is to say dust generated at a playground, say, in the desert southwest versus a pretty wet area?

DR. BECK: In terms of what you might get in airborne?

DR. MUSHAK: Children inhaling, say, airborne chromium as much as arsenic.

DR. BECK: You know that's something -- I could go back. In the model that we use, that's a factor in it for percent ground cover and is directly proportional to the extent of ground covering. And so

what I can do is go back and look at that and see what percent ground cover was used in the model. If you up the ground cover, of course, it will increase it.

Now, our inhalation risks -- I don't have them here. But they were a percent or so of our ingestion and dermal risks. So it would have to be completely arid in order to have anything that I think would be a concern. But I can certainly go back and do that calculation.

DR. MUSHAK: And a final question would be: Could you comment on the difference that, say, John Kissel sees with the defunct copper smelter of Azarko's in Ruston versus where Ed is going with all of his soil studies? It seems to me, if you don't like Ed's soil ingestion choice, just wait a year or two and he'll have something else.

DR. BECK: Although he's kind of honing in around 30, I think, for the Amhurst data. I think one thing to consider, we looked at soil arsenic ingestion at Anaconda. And we used Ed's Anaconda-specific soil ingestion rates, and we used the animal bioavailability studies, and we did a Monte Carlo model in that case.

What we found -- when you're looking at urine, there's two things you need to consider. You're looking at a combination of bioavailability and the combination of soil ingestion. And we did find at Anaconda that we had to either up slightly the bioavailability

estimate or up the soil ingestion estimate for the urine arsenic to match what we calculate the kids should see.

I think it's possible that the Anaconda data -- and, actually, Terry Bower at Gradient is the real expert in this -- may be somewhat of an underestimate and may be a reflection of particle size. We think that the Amhurst data deals with particle size better.

DR. ROBERTS: Dr. Bates, then Dr. Kosnett, then Dr. Kissel.

DR. BATES: Michael Bates. In determining a figure for the bioavailability of arsenic by general uptake, you recommend multiplying a dermal figure, whether it's 3 percent or 6.4 percent, you suggest 3 percent, by the relative bioavailability from ingestion. I was wondering if that could potentially involve sort of counting something potentially twice because the soil will be retarding the absorption of arsenic.

DR. BECK: Well, actually I think you're right. And I put these together. And I think what one needs to do is a ratio of -- I think we have some estimate of what we think fresh soil oral absorption is. And let's say that's 60 percent. So I think going forward, I might consider something more along the lines of 16 percent is to 60 percent as X percent is to say 3 percent.

So I agree with you. I think -- the more I thought about it, I

think there might be some double-counting. I think if you were to do that, if you were to say that the bioavailability of pure soil arsenic, you just add arsenate and give it to the animals, it's about 60 percent. And I think Susan Griffin has some evidence that that may be what you would see. That would increase our dermal estimates by a factor of 1.5. So I think it's perhaps worth looking at.

DR. ROBERTS: Dr. Kosnett.

DR. KOSNETT: I just have a couple of quick questions.

What empiric data did you use to come up with the adherence factors? I notice that you weighted, you know, things by the hands and the whole body and what have you. But what was the underlying empiric data set that you used?

DR. BECK: We relied on the data that are presented in the Exposure Factors Handbook. Our hand-loading we took from Roels, but the other date is in the Exposure Factors Handbook, much of which is derived from studies of Dr. Kissel and his coworkers that have looked at loadings on different body parts under different activity conditions.

DR. KOSNETT: So your approach used Roels's and Kissel's data.

DR. BECK: Yes.

DR. KOSNETT: And you have presented a really comprehensive look at a lot of these issues. But I noticed one -- well, at least one thing that I would like you to comment on that wasn't mentioned. And that is the direct mouth contact with buffer material like wood chips, what have you. What is your response to that as a potential route of exposure?

DR. BECK: Well, I think that one could consider wood chips. But I think what I would do is I would sieve those samples. And then I'd say, when kinds come into contact with wood chips, what's going to adhere to their hands would be the small particle size not a whole chip but finely ground material that's released from those wood chips. I don't think we have the data now to answer what you'd get. But I think what I would recommend is particle-size sieving and using that data.

DR. KOSNETT: Do you think a child, as someone suggested earlier, might pick up a wood chip and put it directly in their mouth? Should EPA consider that?

DR. BECK: Oh, do you mean like an actual chip?

DR. KOSNETT: Yeah, should that be considered a potential route of exposure? I hadn't noted that.

DR. BECK: I think it would be an infrequent occurrence. I

think if you were to look at it, though, you would need to -- now you're talking about bioavailability of a large particle. We looked at bioavailability of sawdust-type material or what Vas looked at was this dislodgeable material.

I think if you wanted to look at something like that, before you would do it, I would recommend that there be some consideration given to the fact that it's a large material and some of it is going to pass through without being absorbed. I think that I would recommend some actual data on bioavailability of large particles if that was something to consider as well as a reduced frequency of uptake.

I mean, it kind of falls into the pica child category where it's an infrequent occurrence. We don't really -- with pica, we don't really have good data on how to estimate it. We tend to estimate it qualitatively. At least in this case, I would consider frequency; and I would want to consider bioavailability.

DR. KOSNETT: Okay. And, finally, when you did the risk assessment -- I just maybe heard incorrectly. I want to make sure -- you said that you used 50 percent as the bioavailability for the dislodged material. Or did you use the 16 percent that you suggested in the beginning?

DR. BECK: Dislodgeable, actually, it was 47 percent. And

then soil, we used 16 percent.

DR. KOSNETT: Thank you.

DR. ROBERTS: Dr. Kissel.

DR. KISSEL: John Kissel. If I understood you correctly, you're ratcheting down the hand to mouth at the quarter of the day for the three hours that the kid is at the playground.

DR. BECK: Right.

DR. KISSEL: Which means that you assume that when the kid leaves the playground his hands are clean; and if so, why?

DR. BECK: I assume that because we know that when kids eat soil that when they're inside they're eating soil that's from the house dust and they're not eating soil from the outside dust. And that's based on the Calabrese studies. I don't know whether it's a function of hand washing or loading and removal, but it's based on the assumption that soil ingestion occurs over the whole day. And when you're eating outside soil, it's outside. And when you're eating dust, it's inside.

DR. KISSEL: I think that all you can conclude from the Calabrese work, if you accept it, is that some portion of the stuff comes from dust and some comes from soil but when those ingestions occur is completely undisclosed by that work. And if you're going to assume that you're down to 20 or 40 square centimeters a day of hand,

that is, that you're harvesting from, then, in fact, the hand could be loaded up and maintain that load until the kid goes to bed or after the kid goes to bed. He could have it the next day. Unless there's a washing event, there's no reason to believe that the hand has gotten clean. And so the kid could, five hours after he has been at the playground, be eating playground dirt off of one of his fingers --

DR. BECK: Right.

DR. KISSEL: -- if it wasn't otherwise removed. I think that I have a problem with that assumption.

DR. BECK: Okay.

DR. KISSEL: I would cut you some slack on another one, which nobody else has brought up, which is all of these dermal absorption numbers are 24-hour numbers. And if you're going to deal with one thing on a time basis, then you ought to deal with other things on a time basis. And there is no real reason to assume that -- well, there should be some temporal distribution of stuff on skin as opposed to just assume that everything is on for exactly 24 hours.

DR. BECK: Right. I think that when you interpret the Calabrese study there must be some washing event that's going on, otherwise I don't think you'd see this difference in soil ingestion as part of house dust versus exterior soil. But I agree that it might be

something that you want to look at in some more detail.

DR. ROBERTS: Dr. Smith.

DR. SMITH. Andrew Smith. Two questions. One is a previous speaker -- I think it was from the Environmental Working Group -- characterized the SCS data as being overly representative of wood products that had either been treated with a sealant or had been treated with some sort of water repellent in the factor. And you mentioned that only one of the products had been treated with a stain sealant, I assume, post- treatment.

Can you just clarify for us of the products that are in the SCS data set to what extent have they been treated either pre the factory or at some point with a repellant versus posttreatment with a sealant.

DR. BECK: Only one of the SCS samples had a true sealant. The way that the study worked is that SCS went out and bought the wood and then treated it themselves except for one sample. And only one of the treatments they used was a true sealant, and that was polyurethane. The others are brighteners and stains, and those are not sealants.

There was one factory applied water repellant that was used which turns out actually had the highest dislodgeable arsenic of all the samples. So it's not correct that they were all sealed. Only one

sample was truly sealed.

And then what I did in my risk assessment was to present the data that was on the slide there which was from CCA-treated wood. No treatment post-purchase. And then in the risk assessment, it was the CCA-treated which had a water repellant applied at the factory, a type of water repellant. It's a pressure type, so you wouldn't be able to apply it yourself.

DR. SMITH: And I don't recall seeing the CSC data in our packets. Do we have that study available to us that would give all the details, both on the study itself and also in terms of the wood products and what they were?

DR. BECK: That data was given to EPA. And I know we're trying -- do we have it?

DR. SMITH: Is that something we can get within the next 24 hours so that we can have a chance to look at it during these deliberations?

VOICE: I'm trying to get it for you in the next hour.

DR. SMITH: That would be great. One last question.

You came to a conclusion that you felt the sealant data was inconclusive, and, therefore, not to be recommended as dealing with the arsenic issue. I think, as you're aware, the last time I looked at the

web pages, a number of the manufactures actually recommend that their wood products be sealed or treated with a sealant every year or two. Can you comment as to why they're making that recommendation because presumably it's doing some benefit to the wood to protect it from ageing and weather and et cetera.

DR. BECK: My understanding, and I hope that I'm -- certainly one of my colleagues in the industry can add to this. It's more for aesthetic purposes. Some of what they're recommending stains and brighteners, so that's not even sealants and that's for aesthetic purposes. Sometimes it's for water repellency so you don't get the water. It's going to reduce cracking. So it's more for aesthetics and function rather than dislodgeable arsenic.

DR. SMITH: Do you know if the industry has any information related to the effectiveness of sealants or any sort of treatment in reducing the cracking of the wood?

DR. BECK: You'd have to ask one of the members of the industry. I mean, tomorrow, I believe we have some time for one of the members speaking so that's something that they can speak to.

DR. ROBERTS: Barbara, I just have a very quick question as a follow-up on an earlier question from Dr. Mushak about the inhalation exposure used. The model, you mention that it factors in vegetative

cover.

DR. BECK: Right.

DR. ROBERTS: Is it the PEF model?

DR. BECK: Yeah, it's one of the standard EPA erosion models.

DR. ROBERTS: I think that that -- just as a brief comment. I think that that model, unless you used a version of it that's specifically for disturbed soils, is for undisturbed soil. And I think that in a playground situation that would certainly qualify as disturbed soils. So you might want to take a look at the inhalation model and be sure that it covers the kind of situation you might see with kids running around and kicking up dust in playground.

DR. BECK: Sure. That's straight forward.

DR. ROBERTS: Dr. Mushak.

DR. MUSHAK: Yeah, one question about the chips versus intact structural pieces. I think one of the concerns is that as a function of overall volume that the amounts of dislodgeables in surface areas with these chips is much higher. So that I think we're not concerned so much that a child may swallow a chip, which I think may have more to do with obstructed airways than perhaps bioavailability; but I think it's a concern that children, over the course of a day, would just keep slurping on these wood-chip surfaces and

thereby release and ingest by direct oral contact a horrendous amount of dislodgeables compared to, say, an intact surface. There's a low surface to volume ratio.

DR. BECK: Are you saying like licking a wood surface?

DR. MUSHAK: No. Sticking a chip -- a child sticking a chip in his mouth, tossing it away, et cetera, et cetera. You know you can get a lot of exposure by the inadvertent contact with something that is not swallowed.

You will recall that Bob Bornshein's studies with the intermountain west lead inferential analyses of blood lead versus exposures. That properties that had a lot of nonbiodegradable cigarette filters, those kids had much higher blood leads than those soils that didn't have discarded cigarette butts. And one logical explanation of that is that these kids just go around slurping on the ends of these cigarette filters. So it could be a medium for transfer rather than a direct GI absorption from a wood chip.

DR. BECK: Are you talking about mulch, or are you talking about a chip of wood coming off?

DR. MUSHAK: Well, mulch, as well as a chip coming off. I think the same principle applies. That when you get less or when you get a surface area to volume ratio that's much higher than an intact

four-by-four beam, say, then I think the potential for an enhanced rate of release per oral activity is much higher.

DR. BECK: You know what I think might be useful -- and I think that John Kissel's comment earlier was very insightful -- is that it might be useful to run through some calculations for that or to run through some calculations, say, with EWG assessment and say what kind of urine arsenic would you be expecting if these events were occurring. And there are a number of urine arsenic studies out there with children. So it might be worthwhile, at least, seeing what you're seeing in the real world.

DR. ROBERTS: Thank you very much, Dr. Beck, for your presentation and answering all of our questions.

We've had some tremendous discussion this afternoon and opportunity to get a lot of great information. Unfortunately, the ability for the brain to sustain activity is finite. I think that one of the things we need to think about is perhaps wrapping up the public comment session for today and beginning again in the morning.

So we have four people listed as public commentors. I know one who has a short presentation will not be here in the morning and has requested the opportunity to go ahead and make their comments now. And I think we need hear what that person has to say.

Let me just ask very quickly if any of the other listed public commentors or people that want to make public comments would not be able to do so if we did this first thing in the morning. Hearing no one, then let's go ahead and extend our public comment period long enough to hear from Bill Walsh from the Healthy Building Network.

Is Bill Walsh here? Great. Would you introduce yourself to the Panel, please.

MR. WALSH: My name is Bill Walsh, and I work with an organization called the Healthy Building Network. And I appreciate you allowing me to go today because I could not be back tomorrow morning.

I'm not a scientist, so you can imagine how riveting this day has been for me. I bring the perspective, however, of parents and consumers who will be looking at the bottom lines or maybe the headlines of your deliberations; and I ask you to bear with me on that.

In this particular case, I think it's very relevant because, for more than a decade, the EPA has chosen to allow the treated-wood industry to self-regulate on this issue. And, therefore, your findings will be primarily communicated to the public by the manufacturers and retailers who sell this product.

And there is a pattern in practice of corporate communications,

a good body of record, that I think as scientists you should be aware of and you should understand how the average person will receive the information that you're receiving today. And with that, if we could go to the next slide.

I'll briefly talk about three basic ways that the public receives information from the treated-wood industry about arsenic-treated wood. And then there's the Consumer Safety Information Program as kind of an aside. Next slide.

If you go to the American Wood Preservers Institute Frequently Asked Questions section of their web site, the question posed is: Is contact health risks for children, and the unequivocal answer is no.

But what I really want to turn your attention to is the very bottom two lines of the slide and in your packet which says, "An extensive 1990 report by the CPSC found that CCA-preserved wood is an appropriate material for playgrounds." This was in a briefing that the AWPI made to the CPSC earlier this year in August. Next slide.

What the CPSC did say in 1990, if you look at that study, there is no finding. There is no suggestion that the wood is appropriate material for playgrounds. There's a very small analysis mostly of wood that had been preserved by what is called a "sealant," the distinctions that Dr. Beck drew.

However, what the executive summary of that memorandum did say was that this suggests a possible hazard might be created when playground equipment is built with unfinished pressure-treated wood from retail sources. And take a look at playgrounds and decks and look at the finished nature of those. Generally, we're talking about unfinished wood.

That study also issued four recommendations for more warnings and safety measures and studies of the raw wood. So once again, that's far different from the assurance that's being given consumers on the web site of the manufactures. Next slide, please.

There's also communication via direct communication in the news media. We have public relations firms here today that are representing the treated-wood industry. And here's a quote from, I believe, litigation under oath that was reported in Florida papers in April of this year, from an industry executive.

"Arsenic is a highly toxic, poisonous, and deadly substance. Womanized (ph) pressure-treated woods does not contain arsenic. Instead, womanized pressure-treated wood contains a preservative formulate by Hickson womanized in wood preservative."

This is what we read in the papers. Next slide, please.

There's more direct communication to consumers via

advertising. These are quotes. The capital letters are theirs, not mine.

"CCA-treated wood is not hazardous, no more acutely toxic to humans than ordinary table salt. Use it for playgrounds. Water from animal troughs made with CCA-treated wood met human drinking water standards."

Next slide.

These are statements that are contained on this multicolored document entitled at the head, "CCA Facts." The next two pictures, very well laid out. And if you look closely, you can see that next to the picture of the playground it says, "Use it for playgrounds." Next to the picture of the picnic bench it says, "CCA-treated wood is not hazardous."

So this is some of the direct communication about the issues we are discussing today that ordinary consumer and parents are getting from the manufactures. Next slide, please.

Same company, Osmose. This is an example of a consumer safety information sheet. I didn't take the color out. There is no color. It's not laid out. The title is not centered. And you can see for yourself, that it's much less appealing nor does it say anything about facts at the top of the statement. Again, quite a mixed message for

consumers and parents. Next slide.

This is an e-mail communication that we have from a very reputable playground manufacturer named Kompan. We're moving now from the manufacturers to the retail communications about the hazards of pressure-treated wood.

The top statement says, "CCA-treated wood is recommended by the Consumer Product Safety Commission CCA-treated wood for preservative, wooden decks, et cetera." The next statement is quite mind boggling. "But there is no scientific or anecdotal evidence of health problems from CCA contact to the users of this products or to the workers who manufacture and install them over prolonged periods of time."

This was written to a parent inquiring about any risks associated with CCA-treated wood in playground equipment. Next slide please.

At the Home Depot, another CCA fact sheet which contains the following language, "EPA approved." Second paragraph, "After years of extensive examination of wood preservatives, the EPA determined that properly used CCA-treated products, including CCA-pressure treated wood, are relatively harmless to humans, animals and the environment. EPA requires no sealers be applied to

CCA-pressure-treated wood for either interior or exterior application. However, See protection."

Which goes to Dr. Smith's point a little earlier that protection is for protecting the integrity of the wood against warping and splitting. Next slide.

This is the full fact sheet. If you just look at the headers, they read as follows:

"Facts: EPA approved; Advantages; Applications and Uses; Standards and Approvals; Durability, Protection." The impact of this is far different from any kind of warning or caution to the ordinary user.

Next slide, please.

On a wall in a Home Depot in Michigan earlier this month, a citizen snapped this picture. "CCA-treated lumber is safe," is what you can see.

The first quote says the following, quote:

"Based on our evaluation, EPA has no risk concerns to public health, even children, from the use of pressure-treated wood. U.S. Environmental Protection Agency." Then various other authorities are quoted on this document. The final quote, "Safe and effective for over 60 years."

Oddly enough, if you're not buying treated wood at Home Depot or you're buying plastic fencing you might see the following. Next slide, please.

Turn your attention to the right-hand side of the slide where we've blown up the details on the ranch post and lattice-top fence at the very bottom. It says, "This is environmentally friendly. No arsenic, creosote, et cetera, which can be harmful to children and animals."

Now, this is the same retailer who said that the EPA had determined that this was relatively harmless. So if you're in the wood department dealing with arsenic, you're reassured. If you're in the plastic department, you're warned about the wood. Next slide, please.

Material Safety Data Sheet from Hickson. Next side, please.

"Ingestion: Not expected to be a problem. However, see notes to physician. Approximately 2.5 ounces, 6 cubic inches, of treated wood dust ingested by a small child may be life threatening."

This is what you get if you're working on a job site maybe. But the average dad going to build a playground doesn't get this information anywhere at the Home Depot. Next slide.

Just a little bit more. Safety information that you will find on the MDSD that is not on the Consumer Safety Information Sheet.

Quote, "Avoid frequent or prolonged contact with the skin"; quote, "This product should not come in contact with food or feed." Yet we have picnic benches being sold with it.

Quote, "Individuals with preexisting disease and/or a history of ailments involving the skin, kidney, liver, respiratory tract, eyes, or nervous system, are at greater risk than normal risk at developing adverse affects from woodworking operations with this product."

Again, this is what the professionals might get from the MDSD sheet, but none of this information is transmitted to consumers or parents relative to the advertisements in the reassurances they're receiving.

Absent some known benefit from arsenic, why should children be subjected needlessly to any degree of risk from arsenic on their playthings when it is so entirely avoidable. Right now the very companies that manufacture the arsenic treatment, manufacture and market abroad as safer arsenic-free compounds. They're are comparably priced; they perform comparably; and, indeed, in some sections of this country, if you go to a lumberyard and buy pressure-treated wood, you're getting arsenic-free wood. They're not even telling you. It's the same price. They just sell it as the topical product pressure-treated wood.

So some consumers are being protected without even knowing it, depending on the retailer they chose.

And what's happening with the EPA program right now is that we're shielding the laggers in the industry and we are building a market barrier to the leaders in the industry who want to do the transition at the expense of concerned parents and their children.

This is a risk in a world where risks, we're always told, it's the mantra, risks cannot be completely avoided. We got one here. And I hope you consider that as you continue your deliberations. Thank you very much.

DR. ROBERTS: Thank you. Are there any questions for Mr. Walsh? Yes, Dr. Morry.

DR. MORRY: Steve Morry, California. The last slide, this safety information on the MSDS, the last item seems to when it says individuals with preexisting ailments and all these categories, and then it says, may have more than normal risk in woodworking operations with this product.

I guess that's a route of exposure that we haven't talked about today. And that is if people buy this pressure-treated wood at Home Depot or wherever and take it home and they're working with their saw and whatever, they're stirring up a lot of sawdust and there's

going to be a potential for some inhalation exposure to the parents who are working with this and to the children if the children are hanging around while the parents are using this.

So I wonder if this is a route of exposure that should also be considered.

DR. ROBERTS: Okay. Any other comments? I guess we're sort of posing that as a question. Yes, Dr. Smith.

DR. SMITH: Andrew Smith, Maine Bureau of Health.

A question for you. I'm interested in your opinions on the use of sealants on existing structures. As you know, aside from the issue of future use, we have many, many CCA-wood structures already out there.

So the question is: What, if anything, can we give for advice to those people? That may be a question that's more relevant for some of our state health folks than it is for the Agency looking forward.

I'm curious, have you looked at the information at all; and do you or your organization have a position on the use of sealants?

MR. WALSH: We look at the information, and we find it very unsatisfactory and not very clear in terms of what to tell consumers. And, in fact, we started with the position that Ms. Beck articulated here which is that most of these things are not really sealants. That is

a very loosely used term. And from what I've seen, you can't rely upon the stains and brighteners to inhibit the arsenic releases.

So what we tend to do is advise people is that a truly impermeable barrier, if you're using polyurethane or perhaps a Latex paint, at least you have an impermeable barrier and you can observe it when it fails, as opposed to the oil-based stains and brighteners that I think give more reassurance than is warranted by anything I've seen. So that's what we tell folks.

In response to the earlier comment, I obviously think that is a route of exposure. And we have been called by people who actually woodwork in confined spaces in their garage. People do not realize that there's arsenic in this wood whatsoever. I didn't until 18 months ago. And so you have these incredible routes of exposure where people would woodwork in their garage, building a picnic table during the winter for use in the summer with pressure-treated wood, that ought to be investigated.

And as for the sealants, that's all we can tell them.

DR. ROBERTS: Okay. I'm sorry, Dr. Morry, I moved on before you got a chance to get an answer to your question. And I think I know the answer, but Mr. Cook or someone else from the Agency could clarify whether that's a kind of scenario that might be covered

down the road.

VOICE: EPA is planning to do that risk assessment and a larger risk assessment. We will address it.

DR. ROBERTS: Dr. McDonald.

DR. MCDONALD: Peter McDonald.

I was wondering if anyone could verify that the Agency was quoted several times in the advertising. Are those quotes appropriate and correct?

MR. COOK: Some of them I think are, but I don't believe all of them are. I'd have to look at the actual pieces of paper. Because there was a consumer information sheet, which I don't have with me, which has the actual language; and we can bring that tomorrow. We have to go back and get it.

DR. ROBERTS: Okay. Thank you. Are there any other questions for Mr. Walsh? Yes, Dr. Wargo.

DR. WARGO: It's actually a question that was brought up by your comments; and it's directed to EPA.

Do you regulate claims of safety or claims of risks in any products that contain CCA?

DR. EDWARDS: I'm not exactly sure what you mean.

DR. ROBERTS: I'm sorry. Could you identify yourself for the

record.

DR. EDWARDS: I'm Debbie Edwards from the --

DR. WARGO: Let me rephrase it, then.

Do you restrict what people can say about claims of safety, or do you demand that products be labeled in a way that warns the public about threats?

DR. EDWARDS: That's a little bit difficult question to answer for pressured-treated wood. Actually, we do carefully regulate those claims on actual pesticides products. They have to be registered. Treated articles, which treated woods is a treated article under our regulations, is exempt from the requirements of FIFRA.

So all of the labeling that you see and all of the things we've been working with industry on to improve the consumer safety information sheet and so forth is a voluntary program.

DR. WARGO: Thanks.

DR. ROBERTS: Any other questions? If not, thanks very much, Mr. Walsh, for your comments. Oh, I'm sorry. Yes.

DR. LEIDY: You might want to --

DR. ROBERTS: I'm sorry. You're going to have to identify yourself.

DR. LEIDY: I'm sorry. Ross Leidy from N.C. State.

You might want to look at the epoxy based resins studies that were done in the '80s by Brady and his group at Georgia that found that where polyurethanes would eventually allow breakthrough of trimiticides like chlordane and chlorpyrifos and the epoxy based resins and so forth are much better at that preventing breakthrough of these types of compounds.

DR. ROBERTS: Thank you for that point. And thanks very, Mr. Walsh, for your comments.

MR. WALSH: Thank you for the time.

DR. ROBERTS: It's been a long but, I think, productive day. I appreciate the cooperation of the remaining public commentors and their willingness to give us their comments tomorrow morning. We'll try to get to those first thing.

We will reconvene tomorrow morning at 8:30. The Panel I would ask to meet in closed session to cover a few procedural things at 8:15 in our meeting room. So could all the Panel members please meet at 8:15, and we will be resuming our open session at 8:30. Thank you.

[Meeting adjourned at 6:50 p.m.]

CERTIFICATE OF STENOTYPE REPORTER

I, Jane F. Hoffman, Stenotype Reporter, do hereby certify that the foregoing proceedings were reported by me in stenotypy, transcribed under my direction and are a verbatim record of the proceedings had.

_____

JANE F. HOFFMAN

-oo0oo-

I-N-V-O-I-C-E** ****I-N-V-O-I-C-E****

JANE F. HOFFMAN
849 S. Irving Street
Arlington, VA 22204
SS#305-52-5988
TODAY'S DATE:
DATE TAKEN: 10/23/01
CASE NAME: FIFRA Conference
PAGES: 307
LOCATION OF DEPO: Crystal City, VA
COPY SALES TO: N/A
DELIVERY: **CONFERENCE RATE -- 111 PAGES AT EVENING RATE**